

# The assessment of concussion recovery using electroencephalography

by  
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April 2019

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## Abstract

Sport-related concussions are a common injury affecting many players. If a concussion is not properly managed players may return to play before they have fully healed from their injury. This may cause a repeated injury, which can have serious long-term consequences. A need exists for a low-cost method of monitoring post-concussion electroencephalography (EEG) variables to determine when players (especially at amateur level) are ready to return to play. EEG is a promising tool for use in concussion management because of its high temporal sensitivity and ability to detect deficits at the brain functional level. The main aim of the project was to develop a concussion management protocol which could be used to compare different EEG measuring devices. Additionally, the protocol was to be validated by repeat testing of concussed participants.

A protocol was developed to monitor Event Related Potential (ERP) and power spectrum changes post-concussion through EEG measurements obtained from an eyes-closed resting state reading, an eyes-closed tandem stance reading, and two Stroop tests (one naming words and one naming colours). The developed protocol was used to compare a low-cost, portable EEG device (Emotiv Epoc+) to a research-grade EEG device (Brain Products Brainvision), and the data was analysed using averaged graphs and paired observation t-tests. The machine comparison test involved 21 participants who were tested with both devices on the same day. Additionally, the protocol was used to monitor the recovery process of a concussed participant at one, fourteen and 38 days post-concussion with the Brain Products device to aid in the validation of the protocol for concussion recovery management.

The results indicated that the Emotiv ERP graphs compared well to the Brain Products ERP graphs in P300 and N200 peak shapes, but that these peaks had lower amplitudes and a delayed latency by approximately 200 ms. The ERP investigation proved to have low statistical power, and should be repeated with more participants. The power spectrum analysis yielded statistically significant differences between the two devices in some areas.

The results obtained from the concussed participant (using the Brain Products device only) showed an increase in P300 and N200 amplitudes as time since injury increased. This is consistent with previous research, and serves to support the notion that the developed protocol is sensitive to changes post-concussion. This should be confirmed with more concussed participants.

Although the trends obtained using the Emotiv device were similar to those obtained by the Brain Products device, high variability of data may pose problems for individual assessment using this device. The Brain Products device was able to detect post-concussion trends consistent with literature using the proposed protocol, but further research is required to confirm these findings.



## Opsomming

Sportverwante harsingskudding is 'n algemene besering, wat baie spelers beïnvloed. Indien harsinningskudding nie behoorlik bestuur word nie, mag spelers terugkeer na die spel voor hulle te volle herstel het van die besering. Dit kan 'n herhaling van die besering veroorsaak, wat tot ernstige langtermyn gevolge kan ly. 'n Behoeftes bestaan vir die ontwikkeling van 'n goedkoper metode om elektroensefalografie (EEG) veranderlikes na harsingskudding te monitor, en om so te bepaal wanneer spelers (spesifiek op amateursvlak) gereed is om na die spel terug te keer. Die hoofdoel van die projek was om 'n harsingskudding-protokol te ontwikkel wat gebruik kan word om verskillende EEG-meetinstrumente te vergelyk.

'n Protokol is ontwikkel om Gebeurtenis Verwante Potentiale (ERP) en krag spektrum veranderinge na harsingskudding te monitor. Dit is gedoen deur EEG opnames te maak van die deelnemers in 'n oë-geslote russtaat, 'n oë-geslote tandem houding, en twee Stroop toetse (een waar woorde en een waar kleure uitgesien word). Die ontwikkelde protokol is gebruik om 'n lae-koste, draagbare EEG toestel (Emotiv Epoc+) met 'n navorsing-gegradeerde EEG toestel (Brain Products Brainvision) te vergelyk, en die data is geanaliseer deur gemiddelde grafieke en gepaarde waarneming t-toetse te gebruik vir die 21 deelnemers. Daarbenewens was die protokol gebruik om die herstellingsproses van 'n deelnemer met harsingskudding met die Brain Products toestel te monitor op een, veertien en 38 dae na die besering opgedoen is.

Die resultate dui aan dat die Emotiv ERP grafieke goed vergelyk met die Brain Products ERP grafieke in terme van die P300 en N200 piek vorms, maar dat die Emotiv pieke laer amplitudes en 'n vertraagde latensie van ongeveer 200 ms getoon het. Die ERP ondersoek het 'n lae statistiese krag bewys, en behoort met meer deelnemers herhaal te word. Die krag spektrum analise het getoon dat statistiese beduidende verskille tussen die twee toestelle in sekere areas bestaan. Die resultate wat van die deelnemer met harsingskudding verkry is toon 'n verhoging van P300 en N200 amplitudes met die verloop van tyd na besering. Hierdie bevindinge stem ooreen met vorige navorsing in die veld, en ondersteun die idee dat die ontwikkelde protokol sensitief is tot verandering na harsingskudding. Hierdie moet met meer deelnemers met harsingskudding bevestig word.

Hierdie studie het belowende resultate getoon vir tendense wat met die Emotiv toestel verkry is in vergelyking met die Brain Products toestel, maar hoë veranderlikheid van die data mag probleme veroorsaak in terme van individuele assessering met die toestel. Die Brain Products toestel was in staat om tendense in die data van die deelnemer met harsingskudding te vind wat ooreenstem met vorige navorsing, maar verdere ondersoek is nodig om hierdie bevindinge te bevestig.

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## Table of Contents

<b>Abstract</b> .....	<b>iv</b>
<b>Opsomming</b> .....	<b>v</b>
<b>Acknowledgements</b> .....	<b>vi</b>
<b>List of figures</b> .....	<b>ix</b>
<b>List of tables</b> .....	<b>xi</b>
<b>List of symbols</b> .....	<b>xii</b>
<b>1. Introduction</b> .....	<b>1</b>
1.1. <i>Background</i> .....	1
1.1.1. Concussions and return to play.....	1
1.1.2. Electroencephalography for concussion management .....	2
1.1.3. Problem statement .....	2
1.2. <i>Motivation</i> .....	3
1.3. <i>Objectives</i> .....	3
1.4. <i>Scope</i> .....	4
<b>2. Literature review</b> .....	<b>5</b>
2.1. <i>Electroencephalography</i> .....	5
2.2. <i>Anatomy and physiology</i> .....	7
2.3. <i>Methods of concussion detection</i> .....	9
2.4. <i>A history of sport-related concussion studies</i> .....	10
2.5. <i>Studies in concussion management and electroencephalography</i> .....	12
2.6. <i>Stroop test</i> .....	16
2.7. <i>The use of the Emotiv Epoc for research</i> .....	20
<b>3. Methods</b> .....	<b>22</b>
3.1. <i>Target population</i> .....	22
3.2. <i>Protocol</i> .....	23
3.2.1. Setup .....	23
3.2.2. Resting state procedure .....	27
3.2.3. Balance test procedure .....	27
3.2.4. Stroop test procedure .....	27
3.3. <i>Data analysis</i> .....	29
3.3.1. ERPs and power spectra of Stroop tasks .....	29
3.3.2. Resting state and tandem stance power spectra .....	30
3.3.3. Responses.....	30

3.3.4.	Filtering .....	30
3.3.5.	Independent Component Analysis .....	31
3.3.6.	Principal Component Analysis.....	32
3.3.7.	Statistical analysis .....	33
<b>4.</b>	<b>Healthy participants: Results and preliminary discussion.....</b>	<b>35</b>
4.1.	<i>Re-referencing and sampling rates</i> .....	35
4.2.	<i>Overall comparison</i> .....	37
4.3.	<i>Machine order effects</i> .....	46
4.4.	<i>Practice effects</i> .....	49
<b>5.</b>	<b>Concussed participant: Results and preliminary discussion .....</b>	<b>53</b>
5.1.	<i>ERP analysis of Stroop tasks</i> .....	53
5.2.	<i>Power spectrum analysis</i> .....	60
<b>6.</b>	<b>Discussion .....</b>	<b>65</b>
<b>7.</b>	<b>Future recommendations .....</b>	<b>68</b>
<b>8.</b>	<b>Conclusion.....</b>	<b>69</b>
<b>9.</b>	<b>References .....</b>	<b>70</b>
	<b>Appendix A: Ethical approval letters .....</b>	<b>77</b>
	<b>Appendix B: Informed consent form.....</b>	<b>81</b>
	<b>Appendix C: Recruitment flyer .....</b>	<b>86</b>
	<b>Appendix D: Event Related Potential peaks.....</b>	<b>87</b>
	<b>Appendix E: Power spectrum peaks .....</b>	<b>88</b>
	<b>Appendix F: Response times and number correct.....</b>	<b>90</b>

## List of figures

Figure 1: The neuron (Ranjitkar, 2016).....	5
Figure 2: Parts of the human brain (Queensland Brain Institute, 2018).....	8
Figure 3: A page from Stroop's original materials for his experiments, printed in 1932 (John Ridley Stroop Digital Archive, Center for Restoration Studies – Brown Library – Abilene Christian University).....	17
Figure 4: Emotiv Epoc (a) device (Emotiv, 2018) and (b) device mounted on head .....	24
Figure 5: Brainvision (a) device and (b) device on head .....	25
Figure 6: Brain Products impedance scale .....	25
Figure 7: Electrode configuration for (a) Brain Products and (b) Emotiv .....	26
Figure 8: Tandem stance.....	27
Figure 9: Instruction screens for (a) Stroop words and (b) Stroop colours tasks .....	28
Figure 10: Flow diagram of Stroop task .....	28
Figure 11: Stroop test with red, blue and green.....	29
Figure 12: Components of (a) eye movement, (b) channel noise, (c) muscle activity and (d) brain activity .....	32
Figure 13: Re-referencing the (a) Brain Products (shown for Fz) and (b) Emotiv (shown for AF3) Stroop words data .....	35
Figure 14: Re-referencing the (a) Brain Products (shown for Fz) and Emotiv (shown for AF3) Stroop colours data .....	36
Figure 15: Downsampling the Brain Products (a) Stroop words and (b) Stroop colours data (shown for F3).....	36
Figure 16: Brain Products (red) and Emotiv (green) (a) Stroop words and (b) Stroop colours ERP comparison.....	38
Figure 17: Stroop words ERP comparison for (a) Frontal, (b) Parietal and (c) Occipital regions.....	39
Figure 18: Stroop colours ERP comparison for (a) Frontal, (b) Parietal and (c) Occipital regions.....	40
Figure 19: Power spectrum comparison of (a) the resting state data and (b) the tandem stance data.....	45
Figure 20: Machine order effects on Stroop words ERPs for frontal (a) Emotiv first and (b) Brain Products first, parietal (c) Emotiv first and (d) Brain Products first and occipital (e) Emotiv first and (f) Brain Products first regions.....	46
Figure 21: Machine order effects on Stroop words ERPs for frontal (a) Emotiv first and (b) Brain Products first, parietal (c) Emotiv first and (d) Brain Products first and occipital (e) Emotiv first and (f) Brain Products first regions with outliers removed.....	47
Figure 22: Machine order effects on Stroop colours ERPs for frontal (a) Emotiv first and (b) Brain Products first, parietal (c) Emotiv first and (d) Brain Products first and occipital (e) Emotiv first and (f) Brain Products first regions with outliers removed.....	48

Figure 23: Stroop words ERPs from the (a) Brain Products (shown for Fz) and (b) Emotiv (shown for the average frontal region) device split into whether the device was the subjects' first or second mode of testing.....	50
Figure 24: Stroop colours ERPs from the (a) Brain Products (shown for Fz) and (b) Emotiv (shown for the average frontal region) devices split into whether the device was the subjects' first or second mode of testing .....	51
Figure 25: ERP comparison between the first and second (a) Stroop words and (b) Stroop colours tasks shown for the averaged frontal region .....	52
Figure 26: Stroop (a) words and (b) colours Fz ERPs for the concussed participant .....	53
Figure 27: Stroop (a) words and (b) colours Pz ERPs for the concussed participant .....	54
Figure 28: Stroop (a) words and (b) colours Oz ERPs for the concussed participant .....	55
Figure 29: Concussed Stroop words average referenced ERPs for (a) frontal, (b) parietal and (c) occipital regions .....	57
Figure 30: Concussed Stroop colours average referenced ERPs for (a) frontal, (b) parietal and (c) occipital regions.....	59
Figure 31: Resting Power Spectrum for (a) original and (b) pruned data .....	60
Figure 32: Tandem Power Spectrum for (a) original and (b) pruned data .....	61
Figure 33: Concussed Stroop (a) words and (b) colours power spectra.....	64

## List of tables

Table 1: Statistical analysis of overall ERPs .....	41
Table 2: Statistical analysis of overall ERPs with outliers removed.....	42
Table 3: Statistical analysis of overall ERP graphs.....	42
Table 4: Statistical analysis of power spectrum results .....	43
Table 5: Statistical analysis of power spectrum results with mean removed .....	44
Table 6: Average response times (per word/colour) .....	49
Table 7: Statistical analysis of practice effects.....	49
Table 8: Concussed ERP peaks compared to healthy averages for Stroop words [ $\mu\text{V}$ ] .....	57
Table 9: Concussed ERP peaks compared to healthy averages for Stroop colours [ $\mu\text{V}$ ] .....	59
Table 10: Concussed resting state power spectrum peaks per frequency band [ $10 \cdot \log_{10}(\mu\text{V}^2/\text{Hz})$ ].....	62
Table 11: Concussed tandem stance power spectrum peaks per frequency band [ $10 \cdot \log_{10}(\mu\text{V}^2/\text{Hz})$ ].....	62
Table 12: Individual ERP peaks [ $\mu\text{V}$ ] .....	87
Table 13: Resting state power spectrum frequency band peaks [ $10 \cdot \log_{10}(\mu\text{V}^2/\text{Hz})$ ] .....	88
Table 14: Tandem stance power spectrum frequency band peaks [ $10 \cdot \log_{10}(\mu\text{V}^2/\text{Hz})$ ].....	89
Table 15: Numbers of correct responses (out of 36).....	90
Table 16: Average response time per subject per task [s] .....	91

## List of symbols

$A$	Mixing matrix
$B$	Eigenvector matrix of $R_u$
$C$	Result of Principle Component Analysis
$D_u$	Eigenvalue matrix of $R_u$
$n$	Sample size
$N$	Number of recorded signals
$P$	Probability
$R_u$	Data covariance matrix
$S$	Source signal
$t$	Test statistic
$U_u$	Eigenvector/modal matrix
$W$	Separation matrix
$X$	Electrode signal
$\mu$	Mean
$\sigma$	Standard deviation



# 1. Introduction

This chapter introduces the project, including the relevant background surrounding the topic, and describes the motivation, objectives and scope of the project.

## 1.1. Background

### 1.1.1. Concussions and return to play

A concussion is caused by rotational or angular acceleration forces applied to the brain, normally from a blow to the head, and is characterised by symptoms such as a temporary loss of consciousness, amnesia, disturbed vision and nausea (Ropper and Gorson, 2007). Concussions may occur in any sport, but are most common in contact or collision sports such as rugby or boxing. Concussions in soccer are also very common due to “heading”, which is employed up to 800 times by professional players in a single season. This does not include practices or training sessions (Rubin and Ashbaugh, 1999).

Most concussions are thought to go unidentified, yet an estimated 3.8 million sport-related concussions are reported in the United States each year, rendering the concussion one of the most common sport related injuries in younger players (13-15% of all sport related injuries) (Daneshvar, 2011). Khurana and Kaye (2012) indicate that reasons for the underreporting of concussions may include a lack of awareness by the players of the significance and symptoms of a concussion, the pressure and desire to continue playing, and the perception that reporting the concussion would negatively impact the player's or team's professional and financial position.

The cognitive domains normally affected by concussion include attention, memory, higher cognitive abilities such as executive functioning, and information processing (Kosaka, 2006). Most cognitive concussion tests aim to test across these areas for deficits. A common tool that has been used for both side-line assessments and clinical use is the Sport Concussion Assessment Tool (SCAT), the use of which mandates the inclusion of a baseline measure taken when no symptoms are displayed. A return to this baseline measurement is indicative of the player's readiness to return to play. However, before a final decision is made, a more rigorous testing procedure is often considered, examples of which include the immediate post-concussion assessment and cognitive testing (ImPACT), automated neuropsychological assessment metrics (ANAM) and CNS Vital Signs (Slobounov et al., 2012).

Studies have shown that most athletes completely recover with respect to symptoms, cognitive dysfunction and other impairments approximately seven to ten days post-injury. The problem remains pinpointing when recovery has been fully achieved at the brain functional level. Studies have shown that physiological abnormalities can be detected beyond the point at which the subject's symptoms have resolved. This was evaluated using advanced functional neuroimaging techniques (McCrea et al., 2010).

### **1.1.2. Electroencephalography for concussion management**

Studies have used Electroencephalography (EEG) and Event Related Potentials (ERPs) to demonstrate that there is a gap between normal-appearing clinical performance and subtle deficits in cognitive performance. EEG has been used extensively to examine electrical activity associated with normal brain function, but lately ERPs have been used to provide insight into the neural processes underlying perception, memory and action. ERPs have a high temporal sensitivity, but poor spatial resolution. This makes source localization difficult, but ERPs are still useful in concussion testing as they provide insight into select aspects of cognition which may have been altered during a concussion. (Broglia et al., 2011)

Although neuroimaging tools are not commonly used in practice, tools such as EEG have the ability to assess neural physiology while clinical concussion assessment tests are being completed. EEG has also been shown to be a reliable tool in concussion management (Teel et al., 2014). There are limited studies investigating the relationship between concussion and neurophysiology using methods such as EEG (Pearce et al., 2015). However, some of the EEG research suggest that event-related brain potentials (especially P300/P3) are well-suited to identify aspects of cognition that remain dysfunctional for an extended period of time after concussion (Broglia et al., 2011). These studies indicate that subjects with a history of concussions typically display smaller P3 amplitude and longer P3 latency relative to control groups. This suggests deficits in cognitive function in terms of the orientation of attention, cognitive processing speed, cognitive control, and the allocation of attentional resources. It has also been shown that there are deficits across motor potentials, indicating dysfunction of motor control and coordination. These deficits can exist long after individuals are cleared to resume sport participation by traditional assessment methods (Broglia et al., 2011).

Another study (Teel et al., 2014) found that EEG power is significantly decreased in a concussed group compared to normal controls over several testing modalities. Again, it was shown that the concussed participants passed all the traditional concussion assessments, but still showed pathophysiological dysfunction when evaluating EEG variables. This indicates that concussed participants are able to use compensatory brain networks to achieve normal functioning. The findings in these studies further suggest the need for additional research to fully determine the extent to which concussion affects the brain and cognition (Broglia et al., 2011).

### **1.1.3. Problem statement**

In order for EEG methods to be practically implemented for the use of concussion diagnosis and recovery monitoring, the use of low-cost EEG systems should be investigated. Traditional EEG systems are expensive, bulky, not mobile, and require prior training to administer and interpret. If a simple, low-cost and mobile system could prove to have sufficient accuracy to monitor post-concussion changes in EEG variables this would prove a valuable contribution to the sports-

medicine field. The focus of this research will be to develop a protocol based on previous research, to compare the data obtained with a low-cost system to that obtained with a research-grade system for the developed protocol, and to validate the protocol using concussed participants.

## **1.2. Motivation**

Sport players tend to downplay concussions, or not report them, as the competitive sporting environment places pressure on players from coaches, parents and teammates. Players resist being removed from the game as they feel that their team depends on them, they need the exposure of the game, or they are afraid of being replaced. If a concussion is not managed properly a player may return to play while some symptoms or cognitive dysfunctions are still manifesting. A repeated trauma in this case may have serious consequences, as a player is also three times more likely to suffer from a second concussion if they obtained a previous concussion in the same season (Guskiewicz et al., 2003). The effects of a second concussion may include a higher symptom burden and prolonged recovery period (Terwilliger et al., 2016), and may lead to diffuse cerebral swelling, brain herniation and death (Bey & Ostick, 2009).

By increasing understanding of concussions and making monitoring of EEG variables more accessible to all, sports players can be monitored from a young age. If a low-cost device can be proved sufficient for monitoring the recovery of concussions, this would aid with return-to-play decisions in a manner that will increase the safety of these players, as the effect of repeated injury will be minimised. In offering clear, medically supported information about the progress of recovery after injury, this study can aid players, coaches and parents in making smart, well informed decisions about the players' safety. By standardising the protocol of concussion management in amateur settings to a more quantitative approach, external pressures on players to return to play too soon can be minimised.

## **1.3. Objectives**

The aim of this project is to investigate a method of monitoring changes in EEG variables in order to manage pathophysiological symptoms in concussion patients. Post-concussion data should be collected at different points in time post-injury in order to determine at which point readings have stabilised. This will help predict how long athletes should rest before returning to play post-concussion, as well as indicating that the developed protocol is sensitive to post-concussion changes in EEG variables. The use of a low-cost alternative for use in concussion management should be investigated in order to ensure that the developed protocol can be used in amateur environments where funding is often a problem.

The project objectives are as follows:

- Review literature on traditional concussion testing, monitoring of patients using EEG, and existing technology for concussion management.
- Identify the key EEG variables for concussion screening in literature.
- Set up EEG screening protocol to include measurements at different points in time post-injury.
- Use the EEG screening protocol to compare the performance of a low-cost device to a research-grade device.
- Identify key differences between measurements of EEG parameters.

Using the data obtained, determine whether measuring EEG variables using fewer electrode channels is a viable option for quick sideline assessments and management of concussions.

## 1.4. Scope

This study investigates the use of a low-cost machine (Emotiv Epoc) for use in concussion recovery monitoring when compared to a research-grade machine (Brain Products Brainvision). Additionally, the recovery of a concussed rugby player is monitored using data analysis methods (ERPs and power spectra analyses) on the results obtained using the proposed protocol with the Brain Products device.

First, relevant literature on the subject is reviewed, including literature on EEG, the anatomy and physiology relating to concussions, current methods of concussion detection, and the history of investigations into sport-related concussions. A review of studies on concussion management with EEG is also included, along with an overview of the Stroop test and its applications. An investigation into studies done on the use of the Emotiv Epoc for research purposes was also conducted.

Next, the methodology followed by the study is outlined, including the target population and participants, the protocol used for EEG recordings, and the data processing methods used.

The results obtained are split into two chapters and includes preliminary discussions as the results are presented. The first chapter deals with the results obtained from the machine comparison using healthy participants, and the second chapter deals with the results obtained from the monitoring of the recovery process of the concussed participant. Results of an ERP analysis and power spectrum analysis are presented, along with the response data for the Stroop tests.

An overall discussion of the results obtained is included, followed by future recommendations and finally a conclusion.

## 2. Literature review

This chapter reviews the literature associated with the study. Firstly, the use of EEG is discussed, followed by the anatomical and physiological aspects of a concussion. Methods of concussion testing currently in use by medical professionals are presented, as well as an overview of the history of concussion in sport. Finally, previous studies in concussion assessment are discussed, as well as the origin of the Stroop test and the use of the Emotiv Epoc for research purposes.

### 2.1. Electroencephalography

The millions of neurons in the human brain use electrical pulses to communicate with and transfer signals to one another. Figure 1 shows a neuron, with its dendrite structures, which assist in receiving the signals from other neurons (Ranjitkar, 2016). These signals are then transmitted to other structures in the body (Marieb, 2015), and a large amount of electrical activity is produced in the brain when these signals are being transmitted. Medical equipment, such as EEG devices, can detect the electrical activity and measure the levels of activity over areas of the scalp. A brainwave pattern is formed from a combination of electrical activity of the brain and the speed of the different types of brainwaves (Delta, Theta, Alpha, Beta and Gamma waves) (Ranjitkar, 2016).

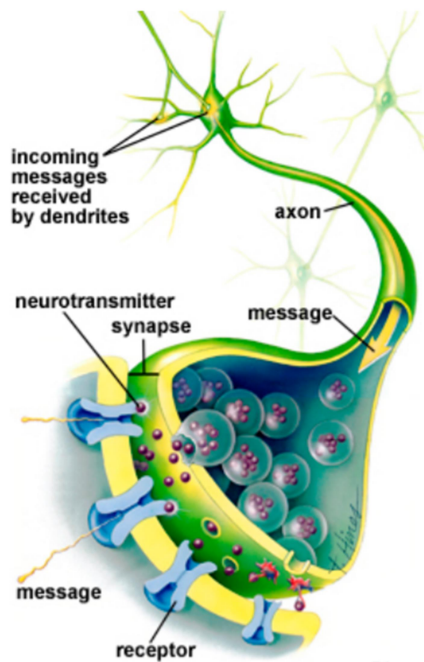


Figure 1: The neuron (Ranjitkar, 2016)

EEG is a graphic representation over time of the voltage difference between two different brain locations (Slobonouv et al., 2012). The signal is generated by cerebral neurons, and is modified by electrically conductive properties of the tissues between the recording electrode and the electrical source, the conductive properties of the electrode, and the orientation of the cortical generator to the recording electrode (Olejniczak, 2006). EEG has a high temporal sensitivity, and is thus highly suitable for examining fast sensorimotor and cognitive functions susceptible to concussive impacts (Slobonouv et al., 2012). EEG is an efficient and fast imaging technique used in the medical science field, and is capable of taking rapid measurements, such that the data on the screen can be seen as a continuous flow of voltage (Ranjitkar, 2016).

Archiniegas (2011) indicates that there are major limiting factors with regards to the capacity of EEG measures in the clinical assessment of concussions. These factors include the lack of control of subjects' homogeneity, the lack of research when EEG assessment was performed, poor experimental designs with respect to collecting of EEG and performance-based assessments, and different time-frames since injury when EEG measures were obtained. A promising measure of sport-related concussion deficits is ERPs, which can provide insight into the neural processes underlying perception, memory and action (Broglio et al., 2011). ERPs are patterns of neuroelectric activation, which occur in response to (or in preparation of) an event. Broglio et al. (2011) reviewed the use of ERPs in sport-related concussion management, and found that ERPs were well-suited for identifying aspects of cognition which remain dysfunctional for an extended period of time post-concussion. These aspects may go unidentified using standard neuropsychological tests, and the chronic effects of concussions remained unclear, despite most injured athletes returning to a pre-injury level of functioning on clinical evaluations within seven to ten days after injury. A gap was identified between normal-appearing clinical performance and covert clinical pathologies which might underlie subtle deficits in cognitive performance.

Broglio et al. (2011) points out important benefits of the ERP approach, specifically its temporal sensitivity and comparability to a large number of reliable and replicable components. However, ERPs have poor spatial resolution, resulting in poor source localisation compared to other neuroimaging techniques. Additionally, ERP signals may be distorted due to the distance of electrodes from the electrical source, and groups of neurons suitable for the recording of ERPs are limited to those near the scalp. The P300, or P3, component (a positive peak occurring at about 300 ms after a stimulus) has captured considerable attention in ERP research, since it is thought to reflect neural activity associated with the revision of mental representation of the previous event. It is thus sensitive to the allocation of attentional resources during stimulus engagement. The P3 has been subdivided into the P3a and P3b subcomponents, which are distinguished by the context in which they occur and their scalp locations. These, along with other components, are thought to provide insight into select aspects of cognition which may be affected by a concussion. For example, it was found that ERP deficits decrease as time from injury increases, and increase as the number of concussions sustained by the individual increases. (Broglio et al., 2011)

Although the P300 component typically appears at approximately 300 to 400 ms after stimulus presentation, it may range from 250 ms to 900 ms in certain cases.

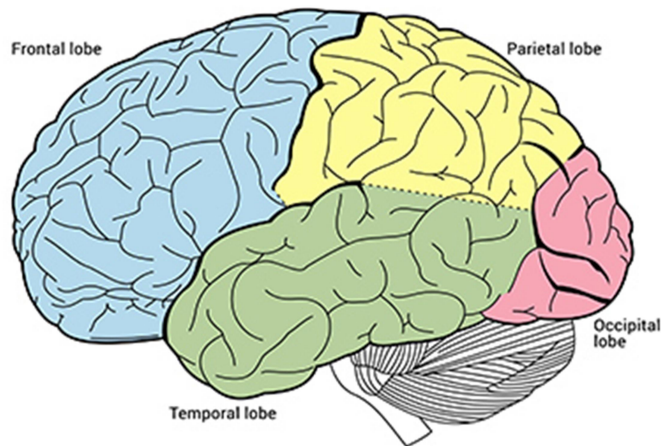
P300 amplitudes generally range from 5  $\mu$ V to 20  $\mu$ V. Broglio et al. (2009) reported significantly larger P300 amplitudes in a control group compared to a group with a history of concussions when performing a novelty oddball task. The control group in this study had an average P300 amplitude of 20.4  $\mu$ V while the group with a history of concussion had an average P300 amplitude of 17.6  $\mu$ V, corresponding to a decrease of 2.8  $\mu$ V or 13.7 %.

Despite these benefits, the shortcomings of the ERP technique for concussion identification were also outlined by Broglio et al. (2011). EEG instrumentation is considered expensive and requires advanced training to collect, reduce and analyse the data. EEG preparation requires a significant amount of time, rendering it difficult to use in an athletic environment since rapid decision making is necessary. A dedicated space, which is free of external stimuli, is necessary in order to minimise noise and maximise signal quality, which may not be readily attainable cost or space wise in most sports settings. These shortcomings of ERP measurement have a major impact on its widespread use, and limit its practical usefulness. A recommendation was made that ERPs should rather be used to make return-to-play decisions, as they had the sensitivity required to identify deficits that would otherwise go unnoticed using routine task performance measures. (Broglio et al., 2011) EEG was the focus of this study, but there are other neuroimaging tools available for use in the examination of sport related concussion. These include functional magnetic resonance imaging (fMRI), magnetic resonance spectroscopy (MRS) and diffusion tensor imaging (DTI) (Slobonouv et al., 2012). Although die diffusion tensor (DTI) can help doctors to predict recovery time for concussed patients, it may require extensive computing power, staff and expertise. The fMRI can provide more insight into pathophysiological mechanisms, but it can be very expensive and require patients to be tested over a long period of time. MRS measures neurometabolites via proton magnetic resonance spectroscopy, and is a non-invasive technique (Pulsipher et al, 2011). These neuroimaging will not be further investigated for the purpose of this study, as EEG shows more potential for low cost, portable options.

## 2.2. Anatomy and physiology

Figure 2 shows an overview of the brain and its different regions. The occipital lobe is responsible for the processing of visual information, and different images can be used to stimulate this part of the brain when using EEG. The parietal lobe is responsible for activities such as interpretation of language, signals from hearing, motor, vision, sensory and memory, but the area of the brain which is mainly responsible for understanding language is the Temporal Lobe. This is also the part of the brain which saves memories from childhood. The Frontal Lobe is responsible for cognitive control, and is responsible for our problem solving, planning, judgements, and intelligence. (Ranjitkar, 2016)





**Figure 2: Parts of the human brain (Queensland Brain Institute, 2018)**

Concussions are thought to be caused by rotational or angular forces to the brain, which result in neural shearing, rather than linear acceleration-deceleration forces which often cause focal macroscopic brain damage (Khurana & Kaye, 2012). This neural shearing is associated with neural depolarization, metabolic derangements at a cellular level and decreased blood flow, and happens to a varying degree among concussions, resulting in different outcomes. (Chadehumbe, 2016) The brief loss of consciousness often associated with concussion appears to be because of rotational forces centred on the midbrain and thalamus which cause a transient disruption of components in the reticular activating system (Khurana & Kaye, 2012).

When a concussion occurs, the mechanical stretch of axons produces injury to the cytoarchitecture and surface architecture of the axon. This leaves the neuron vulnerable to more significant damage upon repeated injury. These forces may also cause short or long term vascular damage (Slobonouv et al., 2012). Although there is no universal agreement on the definition of a concussion, Khurana and Kaye (2012) define a concussion as a “sudden-onset, transient alteration of consciousness due to a combination of functional and structural brain disturbances following a physical impact transmitted to the brain”. They caution against using the term interchangeably with “mild traumatic brain injury” (mTBI), as mTBI can be a serious injury with long term neuropsychological consequences, especially with repetitive occurrences (Khurana & Kaye, 2012).

The effects of mTBI have been linked to several mental health and neurologic disorders later in life, including Alzheimer's disease, chronic traumatic encephalopathy (CTE), Post-Traumatic Stress Disorder, substance abuse, anxiety and depression leading to suicide (Slobonouv et al., 2012). However, Slobonouv et al. (2012) point out that there is conflict among researchers about whether concussion results in long-lasting structural injury to the neuron. Neuropsychologists and other clinical concussion researchers promote the idea that cognitive functional recovery is representative of clinical recovery. This idea is based on data from the restoration of cognitive functioning measured by neuropsychological tests, and not on the healing of the microstructural lesion.



While a study by Bruce and Echemendia (2009) found that there were no short or long term measurable effects of single or multiple concussions on individuals, this is contradicted by other researchers who used neuropsychological testing batteries. These researchers reported abnormalities across multiply concussed groups in the areas of visual and verbal memory (Covassin et al., 2010), delayed recall, executive function (Belanger et al., 2010) and processing speed (Gardener et al., 2010). EEG has been used to identify decrements in the absence and presence of self-reported symptoms and neuropsychological performance in single or multiple concussed cases (Slobonouv et al., 2012). The decrements may be as a result of persistent neurologic dysfunctions from mTBI.

The cognitive domains normally affected by mTBI include attention, memory, higher cognitive abilities (executive functioning) and information processing. MTBI can further cause short term behavioural and affective changes such as depression, anxiety, irritability and compromised social functioning (Kosaka, 2006). Symptoms associated with cases of varying severity of traumatic brain injury (TBI) include headache, fatigue, impaired memory, reduced concentration and attention, reduced information processing capacity, depression, aggression, anxiety, irritability, sleep disturbances and sexual dysfunction (Thatcher et al., 2001). The symptoms of sports-related concussions may include headache, dizziness, nausea, confusion, abnormal balance and postural instability, cognitive defects, sleep disruption and sensitivity to noise and light (Henry et al., 2010). Some of these symptoms overlap with those of TBI outlined by Thatcher et al., and may indicate underlying brain trauma neuropathology at the cellular level (Bigler & Maxwell, 2012) or physiologic damage to neural tissues and its neurometabolic sequelae (Henry et al., 2010).

Due to the varying nature of concussion severity, the recovery process is different for each individual. For cases of sports-related concussion, Chadehumbe (2016) recommends complete rest for 24 to 48 hours post-concussion before returning to learn (i.e. normal academic pursuits). She further recommends at least 24 hours of full physical rest, with an average physical rest of ten to fourteen days before return to play. An athlete should only return to learn when he/she can tolerate 30-40 minutes of concentration, and should only return to play when symptoms fully resolve (Chadehumbe, 2016).

### **2.3. Methods of concussion detection**

According to Slobonouv et al. (2012), allied health professionals had been using the same approach to concussion treatment for nearly 20 years. Clinicians were initially free to choose their own system based on preference. However, this resulted in management and return to play standards that were based on clinical intuition and consensus statements from leaders in the field of neuropsychology instead of on well researched physiologic data (Aubury et al., 2002).

In order to help the clinician determine when an athlete could safely return to play, series testing of patients' cognitive status was recommended in the early 1980s (Hugenholtz & Richard, 1982). Cognitive testing batteries were also introduced to measure a patient's cognitive status. These testing batteries were initially performed with pencil and paper, but have evolved to more easily administered computer-based online batteries. One of the developed tests, the

King-Devick test, is a sideline assessment which takes two minutes. It involves rapid number naming, where the athlete has to quickly read a series of numbers off three test cards. This requires eye movements, vision, language function, and attention (Leong et al., 2015). Worsening performance when compared to baseline scores has been shown to be an accurate indicator of concussion, thus it was investigated by Leong et al. (2015) as a sideline screening tool for concussions since it showed promise as a tool for the assessment of concussions. In a study by King et al. (2015), they noted that the King-Devick test helped identify cognitive impairment in players without clinically observable symptoms. The appeal of the test to them was that it is rapid and easy to administer, as well as that it yields reliable, objective results.

Another common tool used to conduct sideline assessment is the Sport Concussion Assessment Tool (SCAT). This tool requires a comparison of a composite score to a baseline measure, which is taken when the subject is asymptomatic (pre-season). Using the SCAT, a composite score is generated from the Self-Reported Symptoms Score, the Physical Signs Score, the Glasgow Coma Scale, the Sideline Assessment Maddock's Score, the Cognitive Assessment (SAC), and the Coordination Examination Scores (Slobonouv et al., 2012). The SAC includes immediate memory, orientation, delayed recall testing and concentration (Leong et al., 2015). Once the player's SCAT score has returned to baseline and his/her physical symptoms have resolved, a clinician looks to more rigorous neuropsychological testing batteries. These include the immediate post-concussion assessment and cognitive testing (ImPact), automated neuropsychological assessment metrics (ANAM), Headminder and CNS Vital Signs (Slobonouv et al., 2012). These are computer-based neuropsychological test batteries, which evaluate patients across cognitive domains which may include verbal memory, visual memory, executive function, reaction time, cognitive flexibility and fluency. The widely accepted viewpoint across clinical research in sport-related concussion is that the recovery of cognitive detriments and patient self-reported symptoms scores can be used as a measure of clinical recovery from injury (as opposed to functional recovery) (Slobonouv et al., 2012).

## **2.4. A history of sport-related concussion studies**

Gerberich et al. published an article in 1983 which dealt with concussions in high school football. This was the first widely cited article regarding concussions, which found that 14% of the high school football players participating in the study reported a history of concussions (which included a loss of consciousness). Another finding included that 20% of the reported injuries in high school football were concussions. Powell (2001) points out that these results may not accurately reflect recent incidence rates, as a ban was introduced in 1976 by the National Federation of State High School Associations Football Rules Committee. This ban prevented players from using their face mask as an initial point of contact. The players participating in Gerberich et al.'s study had participated in football before the ban, and wore a variety of helmets no longer in use or manufactured. Rimel et al. and Barth et al. also published works in 1983 which identified neurophysiological effects associated with the injury. This increased the discussion around the topic of concussions, and the importance of

the injury was recognised. In the early 1990s, this awareness was spread to the media and fans as a number of high-profile professional athletes retired because of repetitive concussions or post-concussion syndrome, which increased the emphasis on research regarding the identification, management and long-term effects of concussions (Powell, 2001).

The Brain Injury Summit was held in 1994 by the National Athletic Trainers Association Research and Education Foundation. Professionals from neurosurgery, neuropsychology, neurology, sports medicine and athletic training came together to discuss the level of knowledge on concussions, and to make recommendations for future research (Executive Summary, 1994). However, much disagreement still existed on the grading and management of concussions. The main issue with the diagnosis of a concussion is the variety of symptoms with which a concussion may present itself. These symptoms may not be present at all, may disappear quickly, or could linger for an extended period of time (Powell, 2001). In order to better standardise data from different researchers, mild traumatic brain injury (mTBI) was introduced as a synonym for concussion. For an injury to be classified as mTBI, specific criteria regarding the presence of signs and symptoms had to be present (Powell & Barber-Foss, 1999).

In the study by Powell and Barber-Foss, they chose to use mTBI to indicate injuries where the player was removed from participation to be evaluated by a physician and/or athletic trainer for a traumatic brain or head injury before returning to participation. Powell (2001) then pointed out in a further study that with early recognition all concussions can be properly managed, minimising the risks of a negative outcome. He indicated that good management of even the least severe concussions would increase the probability of returning to play without an increased risk of re-injury.

In a study, Lovell et al. (2003) set out to evaluate memory dysfunction and self-reporting of symptoms of concussions in a group of high school athletes. They conducted baseline (pre-concussion) and post-concussion readings at an average of 51.3 days apart for 64 concussed participants. The concussed participants attended follow-up tests at an average of 1.5 days, 4.2 days and 7.2 days post-concussion. They also compared these results to those of a control group consisting of 24 healthy athletes. Their protocol included the administration of the IMPACT computerised neuropsychological test battery, which consisted of seven individual modules which measured aspects of cognitive function (including attention, memory, reaction time and information processing speed). Their results showed that there was a pronounced memory decline post-concussion, even in mild injury cases. This lasted up to seven days post-injury. Self-reported neurological symptoms resolved by day 4, which may be influenced by the expectations of the patient or the hopes of the player for a faster return-to-play. Lovelle et al. (2003) indicated that this emphasized the importance of neuropsychological testing to ensure readiness to return-to-play.

As technology advanced, various studies were conducted which made use of these technologies. A study by Helmer et al. (2014) used Susceptibility-Weighted Imaging (SWI) to study concussion in college ice hockey players. This was the first time that SWI was used to detect symptoms of concussion. It was

also the first time that this method was used to detect changes in the brain over an entire sports season in athletes of both sexes. The study concluded that this method could be valuable in monitoring the players throughout their careers and could help with improved diagnoses and return to play decisions.

A study was conducted in 2018 to prove that concussion loosens insulation around brain cells. Several scans were performed on concussed hockey players, and it was discovered that the protective tissues (myelin) around brain cell fibres were loosened at two weeks after injury. This damage slows the channelling of electrical signals between neurons. MRI imaging (usually performed in hospitals) does not show myelin loosening, which suggests that passing a concussion test may not always be a good indicator if the brain has healed after the concussion (Weber et al., 2018).

Another method of concussion testing, researched by Shahim et al. (2018), involved conducting a highly sensitive blood test to determine when it would be safe for concussed hockey players to return to play. The goal of the study was to compare concentrations in the blood of known biomarkers for concussion. The readings were taken directly after injury, and then monitored over time. This particular study identified a superior blood-based biomarker for assessing subtle brain injury. Shahim et al. indicated that there is no need for a biomarker to make a diagnosis of concussion, as it is usually a clinical diagnosis based on the patient's symptoms. The study specifically focused on a prognostic biomarker which can assist the doctor to determine which players could be at an increased risk of having continuing post-concussive symptoms, and can therefore help to adjust the level of care and rest of the players. Recently, a large number of studies have been conducted using more other advanced technology such as EEG. Some of these studies are discussed in the next section.

## **2.5. Studies in concussion management and electroencephalography**

This section deals with an overview of studies through history which aim to investigate the effects of concussions using EEG and ERPs.

An animal study was conducted in 1973 by Letcher et al., who studied the effects of experimental head injury on the EEG and cortical evoked responses in awake, moderately restrained chimpanzees. This study was the first to investigate these responses in drug-free subjects. In the study, electrodes were constructed by the investigators to measure EEG and implanted under anaesthesia. Holes were drilled in the chimpanzees' skulls to a measured depth, and electrodes were then screwed into the holes. Chimpanzees were reported as free of the effects of drugs within 36 hours, and were administered blows of increasing severity to induce brain injuries. The waking and sleeping EEG and somatic and visual evoked responses (SER and VER) were measured pre-injury to be used as baselines for the 11 chimpanzees. SER are responses which are evoked in brain activity through touch stimuli, while VER are responses evoked in brain activity through images or other visual stimuli. Both SER and VER (measured in  $\mu V$ ) were recorded on magnetic tape and averaged using a computer. The results showed that SER may be a more reliable index of the status of head-injured patients than EEG. (Letcher et al., 1973)

Following this study, less invasive methods of recording EEG were developed, and increasingly many human studies were conducted. Slobonouv et al. (2012) indicate that the most comprehensive EEG study was by Thatcher et al. in 1989, and included 608 mTBI subjects up to 8 years post-injury. The study revealed an increased coherence and decreased phase in the frontal and frontal-temporal regions, decreased power differences between the posterior and anterior regions, and reduced alpha power in the posterior cortical regions. These data trends were confirmed by Thornton (1999).

Thatcher participated in another study (Thatcher et al., 2001) where they conducted an EEG spectral analysis from 19 scalp locations for patients with mild, moderate and severe TBI at 15 days to 4 years post-injury. All patients were in the non-acute or chronic period post-injury at the point of evaluation. They aimed to find a method of determining the severity of a TBI using a discriminant function as an index. In order to determine the severity of TBI, Thatcher et al. mentioned the Glasgow Comma Score (GCS), the duration of loss of consciousness, duration of posttraumatic amnesia, EEG and MRI as options. The GCS was seen as a valuable first observation, but had practical limitations as it was not often measured at the site where the patients were first transported. The duration of loss of consciousness (LOC) was often unknown or unrecorded, and the duration of posttraumatic amnesia (PTA) was also often not obtained in the acute admission stages. At this point, the standard visually read EEG and conventional MRI were not yet sensitive to the differences between mild and moderate TBI, and did not predict outcomes or levels of severity of TBI well. However, quantitative EEG (qEEG) studies had been able to predict severity (and sometimes long-term prognosis) in the range of months to years post-trauma. (Thatcher et al., 2001)

Thatcher et al. (2001) performed a power spectral analysis on 2 to 5 minute segments of eyes-closed resting EEG recorded from 19 scalp locations. Their electrodes were placed based on the 10/20 system of placement, with the left ear lobe as a reference and a 100 Hz sampling frequency. Visible artifacts were removed with the aid of ECG and eye movement electrodes, and the absolute EEG frequency amplitude of the power spectral analysis in the delta (0.5-3.5 Hz), theta (3.5-7Hz), alpha (7-13 Hz) and beta (13-22 Hz) frequency bands were calculated. A discriminate score was then calculated, and they were able to find significant correlations between the EEG discriminant scores, emergency admissions measures and post-trauma neuropsychological test scores.

Furthering the research being conducted with qEEG measures, McCrea et al. (2010) conducted a study on high school and college football players. They aimed to investigate whether a portable, automatic, frontal qEEG acquisition device would be clinically useful in detecting abnormal brain electrical activity following sport-related concussion. McCrea et al. (2010) reported that qEEG studies had indicated abnormalities such as reduced mean frequency of alpha, reduced power in alpha and beta frequency bands, hypercoherence between frontal regions, and decreased gamma frequency in concussed participants (when compared to healthy participants). They limited their montage to frontal scalp locations. The frontal region was considered three times more likely to be affected than other cortical regions as its proximity to bony structures and cavities of the skull makes it particularly susceptible to injury, forming the basis of their

decision. The frontal regions are the most vulnerable for focal deficits after head injury, and the most common symptoms following concussion are as a result of frontal and/or temporal dysfunction (McCrea et al., 2010). Significant differences in qEEG data between baseline data and data obtained at injury and eight days post-injury were found in this study. McCrea et al. (2010) also found significant differences between control and concussed groups at injury and at eight days post-injury, while these differences were not present at baseline or 45 days post-injury. Although concluding that their research expanded the understanding of physiological recovery after sport-related concussion, they indicated that future research was necessary to better understand how qEEG could help the clinical management of sport-related concussion.

Since EEG had been shown to be a reliable and sensitive tool in concussion management, and could help to assess brain physiology while completing clinical concussion assessments, Teel et al. (2014) conducted further research on the use of qEEG. They studied thirteen control and seven concussed participants using 128 EEG channels. Concussed participants were tested within eight days after the injury, and all participants had to complete three assessments. These assessments were an EEG baseline, ImPACT testing, and a Virtual Reality (VR) balance and spatial module. They examined the EEG data using differences in the coherence and power values between the control and concussed groups. Their EEG baseline included two-minute long readings of eyes open sitting, eyes closed sitting, eyes open standing and eyes closed standing. Following this, subjects had to complete a computerized version of the ImPACT tool. Finally, the participant had to stand in the Romberg position while VR equipment made the room appear to sway for thirty seconds. The Romberg position involves the subject standing with one foot in front of the other and hands on hips. The participant also had to navigate through a virtual hallway with the VR equipment. Teel et al. (2014) excluded control participants if they had a concussion history in the past year, known neurological disorders, a diagnosis of attention deficit disorder or any lower body injuries affecting balance. The EEG recordings collected were sampled at 500 Hz and referenced to Cz, and the recordings were notch filtered at 0.5 to 59 Hz and artifacts were manually removed after automatic software identification. They then calculated coherence and power values. (Teel et al., 2014)

This study by Teel et al. (2014) supported the findings of other studies in that they found that EEG reflected concussion differences not seen in clinical measures. Besides the differences found in the EEG data, the only statistically significant difference between the concussed and control groups was in the stationary balance score. They found that EEG power was significantly lower for concussed participants in nearly every task. Coherence values displayed large variances among the different tasks, which the researchers attributed to an attempt by the concussed individuals to recruit additional brain areas to perform the tasks. Teel et al. (2014) concluded that these compensatory mechanisms may not be sufficient to compensate for the lack of power when performing sport which requires high levels of concentration. Returning to play within one week of injury may thus put participants at an increased risk of injury.



However, research indicated that players were eager to return to play, possibly before they were completely healed from injuries. Kroshuis et al. (2015) conducted a study which aimed to quantify concussion under-reporting in a sample of 328 male and female athletes in 19 teams, participating in one of seven sports at four colleges in the United States. They found that more than a quarter of their sample had experienced pressure from at least one source to continue playing after a head impact during the previous year. Furthermore, Meier et al. (2015) found that significantly fewer symptoms were reported by athletes in the acute phase of concussion to team athletic trainers using the ImPACT test compared to symptoms self-reported in a confidential setting using standard psychiatric interviews. These athletes continued to underreport symptoms 9 days post-concussion, after being cleared to play, supporting the notion that the underreporting of symptoms had resulted in the athletes being cleared to play. The symptoms reported in a confidential setting by athletes cleared to play were similar in magnitude to those reported by athletes not cleared to play.

A study by Pearce et al. (2015) indicated that a range of testing modalities should be incorporated rather than one area of measurement in measuring the recovery of concussed patients when making return to play decisions. This was based on their study which found that acutely concussed football players showed abnormalities in motor, cognitive and neurophysiological measures with variable rates of recovery. They also found that neurophysiological measures can provide a valid measure of the underlying mechanisms for neurocognitive and motor responses following concussion.

Hudac et al. (2017) conducted a study with the objective of studying the long-term effects on working memory brain function after a history of concussion in young adults. Evidence was provided of long-term electrophysiological differences after a concussion was sustained, which emphasized the need to gain further understanding of the underlying cognitive and neural mechanisms implicated in concussive injuries. Although there was a lack of behavioural tasks performance differences between the groups, differences were shown in the amplitude and latency of ERP components as well as in neural source activations. Subjects with a history of concussion showed increased P1 and P3 amplitudes, decreased P1 latencies, increased N2 latencies, and different neural source activations for brain source regions critical for working memory, which may suggest an inefficient use of the working memory neural system for a person with a history of concussion. Additional research in this area is therefore required in order to track concussive symptoms to support athlete recovery and improve the overall long-term prognosis.

However, some of the findings of Hudac et al. (2017) seem to contradict the findings in previous research. Broglio et al. (2009) conducted a study on the persistent effects of concussion on neuroelectric indices of attention. The objective of this specific study was to study young adults (with and without a history of concussion) by employing a standard clinical assessment, as well as electrophysiological measures that were highly sensitive to persistent changes in the cognitive functioning. Findings show that there was a larger N2 amplitude for the group without a history of concussion compared to the group with a history of concussion. With regards to the P3 amplitude, it was found that there was a

significantly larger P3b amplitude for the group without a history of concussion than for the group with a history of concussion. Broglio et al. (2009) indicated that these findings correspond with previous research.

## **2.6. Stroop test**

The proposed methodology to follow in Chapter 3 involves a Stroop test. A Stroop test is a test first introduced by J. Ridley Stroop in 1935. His aim was to study interference in serial verbal reactions through a series of experiments. The interference was presented in pairs of conflicting stimuli, namely word and colour stimuli, presented simultaneously (a name of one colour printed in the ink of another colour). The stimuli were varied to maintain the potency of their interference effect- no word was printed in the colour it represents, and words and colours were varied to avoid duplicates next to each other with respect to either rows or columns. (Stroop, 1935)

In the first experiment the subjects were asked to read the words aloud, correcting any mistakes made as they go. The time taken was compared to reading the same words printed in black ink, and it was found that subjects took longer to read the coloured words. The second experiment was similar, except that subjects were asked to name the colours that the words were printed in. The time taken was compared to the time taken to name rectangular blocks of colours arranged in the same way. It was once again found that subjects took longer to identify the colours that the words were printed in. The third experiment dealt with practice effects. After eight days of practice, it was found that the interference was reduced (but not eliminated) in naming the colours, and that the interference was increased when naming the words. One of Stroop's cards used for his experiments is shown in Figure 3.





**Figure 3: A page from Stroop's original materials for his experiments, printed in 1932 (John Ridley Stroop Digital Archive, Center for Restoration Studies – Brown Library – Abilene Christian University)**

The Stroop colour-naming task is considered a classic paradigm, and various versions of the Stroop test have been studied (Ilanand and Pollich, 1999). It is able to illustrate important concepts such as interference and automaticity, and the magnitude of Stroop facilitation and Stroop interference have been observed many times in variations of the paradigm. The first studies on the effects of Stroop paradigm on brain potentials began in the 1980's (Sahinoglu and Dogan, 2016). The study of (Duncan-Johnson et al, 1981) compared the Response Times (RT) and P300 latencies to determine at which stage the interference had occurred. A P300 component was recorded for the congruent, incongruent and neutral conditions, but no differences between the latencies of the P300 were found. However, the RT to the incongruent stimulus was slower when compared with the neutral stimulus. This result shows that the Stroop effect occurred later than the stimulus evaluation.

Aaron and Pollic (1999) furthered this research by conducting a study where manual RT and P300 ERP measures in a Stroop colour naming task were recorded. The P300 was again used to investigate the nature of Stroop effects, and to test the hypothesis that the Stroop effect stems from response- rather than

stimulus-related processes. The findings in this study point out that the P300 latency does not vary in a similar way as RT in a manual key press version of the Stroop colour-naming task, indicating that the Stroop effect does stem from the response-related process, which supports the findings of Duncan-Johnson and Kopell (1981).

In another study, the N400 wave was studied in response to congruent, incongruent and neutral stimuli by using the standard four colours (red, green, yellow and blue) as well as non-verbal signs (X's). The subjects were asked to vocalize the colour of the word, and to name the colour mentally. A conclusion was made that expectancy-induced priming could lead to automatic reading, which facilitated the process of expected targets. (Rebai et al., 1996)

Ilan and Polich (1999) in their study looked at P300 waves from Pz, Fz, and Cz locations in response to congruent, incongruent and neutral stimuli. The names of the colours were used for congruent and incongruent conditions, but for the neutral stimuli they used infrequently used words presented in each of the four colours. This study confirmed that the Stroop effect on RT was created after the evaluation of the stimulus, in later response production stages, after the P300 has been elicited, supporting the research of Duncan-Johnson and Kopell (1981), and that of Aaron and Pollic (1999).

In another Stroop investigation, the subjects were requested to respond with a key press to four colours in congruent and incongruent situations. The coloured X's were used as the neutral stimulus (West and Alain, 1999). As concluded by other researchers, the latency of the response to incongruent stimuli was remarkably longer compared to the congruent and neutral stimuli. Additionally, West and Allain found that there was a difference in the ERP amplitudes over the left parietal regions compared to the right parietal regions with regards to incongruent stimuli, which suggested that the left parietal is not fully active in word-colour conflicts. (West and Alain, 1999)

Lotti et al. (2000) also used the four colours, shown on a dark grey background, in congruent, incongruent and neutral conditions. The neutral stimulus was made up of light grey words. In an interesting variation of normal Stroop tasks, the subjects had to say the colour word aloud in the overt condition, say the colour word silently in the mind in the covert condition and in the manual condition had to give a key-press response. It was found that a strong Stroop-colour word interference was picked up for both vocal versions and the manual version of the test. There were no statistical differences with regards to the RT's of both conditions.

Further studies were conducted by Atkinson et al., Bekci et al., and Zurrón et al., which all confirmed that the Stroop interference arises at the output phase. In the study by Atkinson et al. (2003), the words red, green and blue were used as stimuli, and were written in white fonts in coloured rectangles with a white overall background. The words "low", "case" and "since" were used for the neutral stimuli, and the subjects were asked to give key-press type responses. This study found that stimuli were processed in parallel in a network of brain areas, rather than in a particular structure, and confirmed that Stroop interference arises at the output stage. Bekci et al. (2009) used the Turkish equivalents of the four colour

names, and ERP's from 30 electrode sites were recorded according to the 10-20 system. A key-press response was used, and differences in ERP amplitudes were found which reflected the information processing in relation to the meaning of the stimulus. These findings highlighted both the conflict and the response competition hypotheses. In the study by Zurrón et al. (2009), the Spanish equivalents of the words blue, green, red and grey colours on a black background for both conditions were used. Again, the subjects had to give a key-press response. It was concluded that there was a decrease in ERP amplitudes as a result of the semantic conflict caused by the incongruent stimuli. With regards to the latencies of the peaks, no difference was detected between the two modalities, which could support the conclusion that the processes before the response stage had no influence on the generation of the Stroop effect.

Sahinoglu and Dogan (2016) summarised that most of the studies conducted on the Stroop test used colours as stimuli (red, green, blue and yellow), and used similar electrode locations. All the studies recorded potentials from Fz, Cz, and Pz positions. There are some discrepancies in the studies, for example in the electrode sites used, type of neutral stimuli used, parameters used in the timing of the stimulus presentation, and the response modes that the subjects were required to give. It is, however, important to note that all results confirm that the RT to the incongruent stimulus is longer compared to the congruent stimulus. The ERP's central contribution is to demonstrate the physiological mechanisms underlying the Stroop effect. The shared ERP results show that the latency of the P300 component is not affected by the incongruent stimuli, and it can be concluded that the Stroop effect on RT was created after the evaluation of the stimulus. (Sahinoglu and Dogan, 2016)

Several concussion studies were conducted with regards to an auditory Stroop task. One of the studies was conducted at 4 weeks following a concussion (Catena et al., 2011). Another study's objective was to examine the independent associations between objective dual-task gait balance and neurocognitive measurements during the acute (within 72 hours of injury) and the long-term (2-5 months post-injury) stages (Howell et al., 2018). A study conducted by Howell et al. (2014) attempted to examine how gait balance control was affected by three secondary cognitive tasks with different complexity after a concussion. Another study by Howell et al. (2015) focused on age as a factor that affects recovery after concussion. The dual-task gait balance control deficits after concussion were examined in a group of adolescents and young adults.

Since the Stroop test measures attention and executive functioning (two of the areas identified previously as being affected by concussions), it is ideal for concussion testing. Some studies which used the Stroop test to measure concussion deficits included research on whether obtaining baseline neuropsychological data to assist in the management of sports-related concussion be considered the standard of care (Hinton-Bayre, 2015). Yang et al. (2017) used this to develop a tablet-based software battery (BrainCheck) for concussion detection with regards to sports, emergency department cases, and in a clinical setting. The accuracy of the BrainCheck was evaluated after comparing the tablet-based assessment with physician diagnoses after brain scans, clinical examination and the SCAT3 test. It was found that the tablet-based assessment provided a rapid, portable testing method for TBI.

In another study the Stroop task was combined with the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS) and the Post-concussion Syndrome in order to evaluate the effects of head trauma in college athletes. They concluded that they were limited in their interpretations of the study, but found important implications that emphasized the further exploration in this area of research. (Killam et al., 2005)

## 2.7. The use of the Emotiv Epoc for research

Several studies have been conducted on EEG-based Brain-Computer Interface (BCI) systems. Medical-grade (and research-grade) EEG devices are usually very expensive for end-users, and a few low cost alternatives have appeared on the market. An interesting study on the performance of the Emotiv Epoc headset for P300-based applications was conducted by Duvinage et al. (2013). This study was a quantitative comparison between the low-cost Emotiv Epoc headset and the ANT medical/research EEG device based on a standard P300 Brain-Computer Interface. The objective was to gain a better understanding of the Emotiv Epoc and to be able to supply relevant information to researchers and other users.

In this study the Emotive headset performed remarkably worse than the medical device with effect sizes that varied from medium to large. The Emotiv headset also showed higher relative maintenance and operational costs compared to that of the medical-grade competitor. However, a conclusion was made that the Emotiv Epoc is able to record EEG data in a satisfactory quality level, but should only be selected for non-critical applications. It was recommended that much research should still be conducted, specifically for critical applications. (Duvinage et al., 2013)

Byron et al. (2018) published an article on brain monitoring devices in neuroscience clinical research. The focus was on the potential of remote monitoring by making use of mobile devices, sensors and wearable devices. They found that little prior research had been conducted to test the reliability of these systems for ERPs. However, the existing research shows promise in using these techniques.

One such research study focused on the validation of the Emotiv Epoc EEG gaming system for measuring research quality auditory ERP's. It was concluded that the gaming EEG system may be a valid alternative to laboratory ERP systems in order to record reliable late auditory ERP's (P1, N1, P2, N2 and P3), specifically over the frontal cortices. (Badcock et al., 2013)

Badcock et al. (2015) went on to conduct another study on the Emotiv Epoc EEG system in respect of the research quality auditory event-related potentials in children. Since the previous study proved that the Emotiv Epoc can be adjusted to provide valid auditory ERPs in adults, the study set out to prove that it was also true for children. It was found that an adapted Epoc Emotiv EEG system can be used to record children's late auditory ERP peaks and their ERP components. Badcock et al. reported P300 insignificant amplitude differences between the Epoc and a research-grade device of  $0.16 \mu\text{V}$  for the F3/AF3 electrodes and significant delayed latencies of 15 ms for the Emotiv device at

both the F3/AF3 and F4/AF4 electrode sites. They also reported that, although still adequate for their research, the number of epochs rejected for the Emotiv was significantly higher than the number rejected for the research-grade device. This is an indication of the high variability of Emotiv data.

Maskeliunas et al. (2016) evaluated two low-cost consumer-grade EEG devices, namely the Emotiv Epoc and the Neurosky MindWave. The Emotive Epoc device showed a recognition accuracy of more than 75% with regards to eye blink recognition. This device also performed better for tasks that required concentration and relaxation of the subjects. A conclusion was made that the Emotive Epoc could be more suitable for control tasks when using the level of eye blinking than the other device.

Another research study by Taylor et al. (2016) focused on the evaluation of the Emotive Epoc Brain Computer Interface (BCI) for the detection of mental actions. The focus was specifically on the use of the Emotiv Epoc by normal, healthy users. The results showed that the Emotiv Epoc system serve as a BCI with an acceptable level of accuracy. The system was able to correctly identify mental actions 87.5% of the time.

These studies on the use of the Emotiv Epoc for research applications show promising results for its viability as a low-cost alternative to traditional EEG devices. Further research in this field is necessary to determine the extent of its capabilities.

## 3. Methods

This chapter outlines the methods used in the study. The target population is identified, followed by an outline of the protocol and the data analysis procedures.

### 3.1. Target population

The target population for the study was newly concussed rugby players ranging in age from 18 to 23. Players were included if they could be recruited within 24 hours of obtaining a concussion. The exclusion criteria included a history of mental illness, open wounds on the subject's head, colour blindness and any lower body injuries which may affect the subject's ability to perform a balance test. Maties Rugby, associated with Stellenbosch University, has over 1300 registered players (Maties, 2017), with between 250 and 300 reported cases of concussions each year (Stassen, 2017). This corresponds to a concussion rate of 19-23%. Previous studies investigating correlations between concussion recovery and EEG had sample sizes of about 7 concussed subjects (Teel et al., 2013). It was therefore hoped that a sample size of between 10 and 20 concussed participants could be obtained. Ethical approval was obtained for the study from the Health and Research Ethics Committee (Appendix A).

The study was conducted with the help of Campus Health Services. Concussed rugby players who reported to Campus Health post-injury were asked to participate by the doctor on duty and handed a flyer (Appendix C) to invite them to participate in the study. If they were willing to participate, the principle investigator was to be contacted. Rugby players who are injured on the field with suspected concussions are immediately pulled off the field as per Maties Rugby protocol. These players are then flagged and have to report to Campus Health in order to be cleared. If they are not cleared by a doctor from Campus Health Services, they are not allowed to return to practice or play. Participants who reported with concussions were asked to give preliminary informed consent, which was confirmed by them once they were asymptomatic and their brains had healed from the injury. Consent could be withdrawn by the participant at any time, at which point their data would be disposed of, and they would be removed from the study (Appendix B).

Only one concussed participant was recruited. The participant was a white male rugby player of age 20, and performed the protocol with the Brain Products Brainvision device. The concussed participant was tested at one, fourteen and 38 days post-concussion. This low sample size can be attributed to unforeseen circumstances of a drought in the area, resulting in the cancellation of Rugby in the region for the duration of half the normal season. Additionally, players seem hesitant to respond to flyers, so alternative recruitment methods should be considered for future studies.

A second group was targeted in order to measure the performance of a low-cost EEG device (Emotiv Epoc+). The low-cost device was selected for its lightweight, portable nature and low set-up time when compared to the Brain Products device. This makes the device attractive for use in non-professional athletic environments where costs, space and training of technicians may pose

limitations to the use of EEG for concussion recovery applications. For these tests, 27 willing participants were recruited. Inclusion criteria included no diagnosed concussions within three months of testing and no history of mental illness, while subjects were also excluded if they suffered from colour-blindness, injuries which impaired their balance or open wounds on their heads. These participants were asked to perform the research protocol twice, once wearing the low-cost Emotiv Epoc+ and once wearing the Brainvision EEG machine by Brain Products. The data of five of the subjects was discarded due to recording errors. The Stroop words data (described in Section 3.2) was also discarded for one other participant, but the participant's other data was included. Of the 22 included healthy participants, 12 performed the protocol with the Emotiv device first and 10 performed the protocol with the Brain Products device first to avoid the order of recording from skewing the results.

Of the included healthy participants, 16 were male and 6 were female. The participants were evenly distributed between the test groups with respect to sex. With respect to race, 17 participants were White, one Black, two Coloured and two Indian. The participants ranged in age from 21 to 28 years.

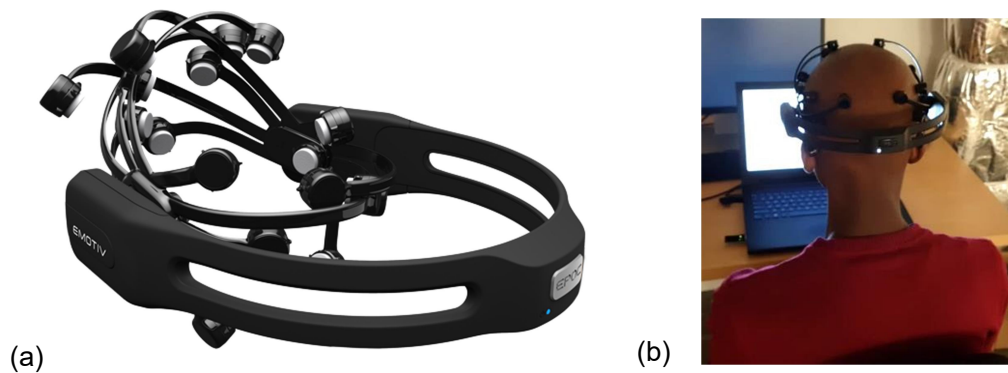
## **3.2. Protocol**

Subjects who gave informed consent were tested using the outlined protocol.

### **3.2.1. Setup**

The first EEG measuring device used was the Emotiv Epoc+, and is shown in Figure 4(a). This device was used with Emotiv Xavier Pure recording software. The wireless device consists of a configuration of 14 electrodes which were saturated with a saline solution before the device was placed on the participant's head. The device was positioned by placing the reference electrodes on the mastoids and the front two electrodes approximately at the hairline (three fingers above the subject's eyebrows). Figure 4(b) shows the device fitted on a person's head. The device measured EEG activity at a sampling frequency of 128 Hz, and wirelessly transmitted the data to a laptop used for recording. The setup time for the Emotiv device was five minutes and could be done without advanced prior training. After recordings were taken, the device was wiped down with a cloth before being packed away. Markers to signify events representing to onset of stimuli for the Stroop tests were sent from the stimulus-producing software to the recording software through the use of a virtual serial port. This method may produce a variable delay in markers, and should be replaced by a more trustworthy method in future research. One such method which warrants further investigation is proposed by Badock et al. (2013).



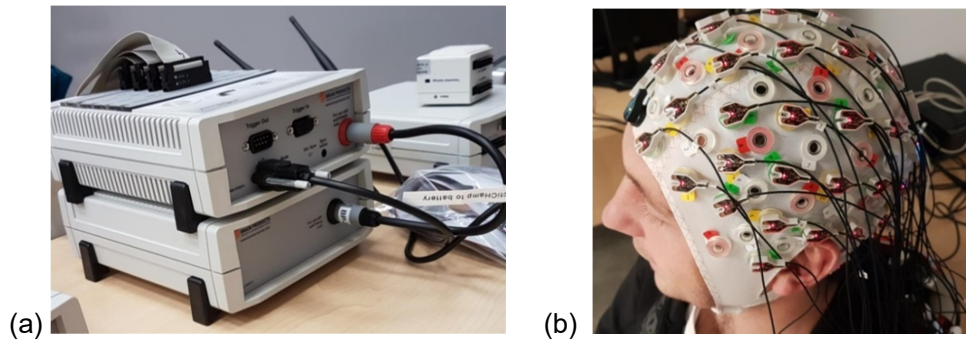


**Figure 4: Emotiv Epoc (a) device (Emotiv, 2018) and (b) device mounted on head**

The second EEG measuring device used was the Brainvision by Brain Products (shown in Figure 5(a)), which is an expensive machine acquired by the Stellenbosch University Neuromechanics Lab for research purposes, and served as the standard to which the Emotiv device was compared. This device has been used previously as the standard to which to compare other EEG devices (Krigolson et al., 2017). Recordings from this device were obtained using the Brainvision Recorder software. This device was used with 64 channels for healthy participants and 32 channels for the concussed participant. High setup time was required for this device, with the preparation time for 64 channels set at approximately one hour and the preparation for 32 channels taking 30 minutes. Since the required time was considered a major factor for athletes when deciding whether they were willing to participate in the study, a decision was made to only use 32 channels for the concussed participants in order to minimise the time required for testing and hopefully recruit more participants in this way. The high setup time is attributed to the fact that device's electrodes had to be gelled with a conductive gel to facilitate measurements. In addition to the high setup time, the Brain Products device also required high cleanup time of about 40 minutes, as the gel had to be washed off the cap and each individual electrode after recording. Markers for the appearance of stimuli were fed from the stimulus-producing computer to the recording computer via the use of a parallel port (hardware triggering). This is a more reliable method than that used for the Emotiv device with a small, standard delay.

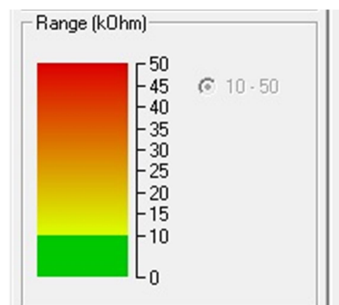
The subject's head was first measured to determine the cap size used, and the relevant cap was fitted without electrodes to ensure that it was not too loose or too tight (especially for participants who measured between cap sizes). This cap size corresponded on the circumference of the subject's head (in cm), and caps were available in sizes 54, 56, 58 and 60. The cap and electrodes were then assembled separately and placed on the subject's head, as shown in Figure 5(b). The cap was positioned using a measuring tape so that the reference electrode (Cz) coincided with the middle of the subject's head (measured from ear to ear across the head and from the middle of the eyebrows to the occipital bone across the head).





**Figure 5: Brainvision (a) device and (b) device on head**

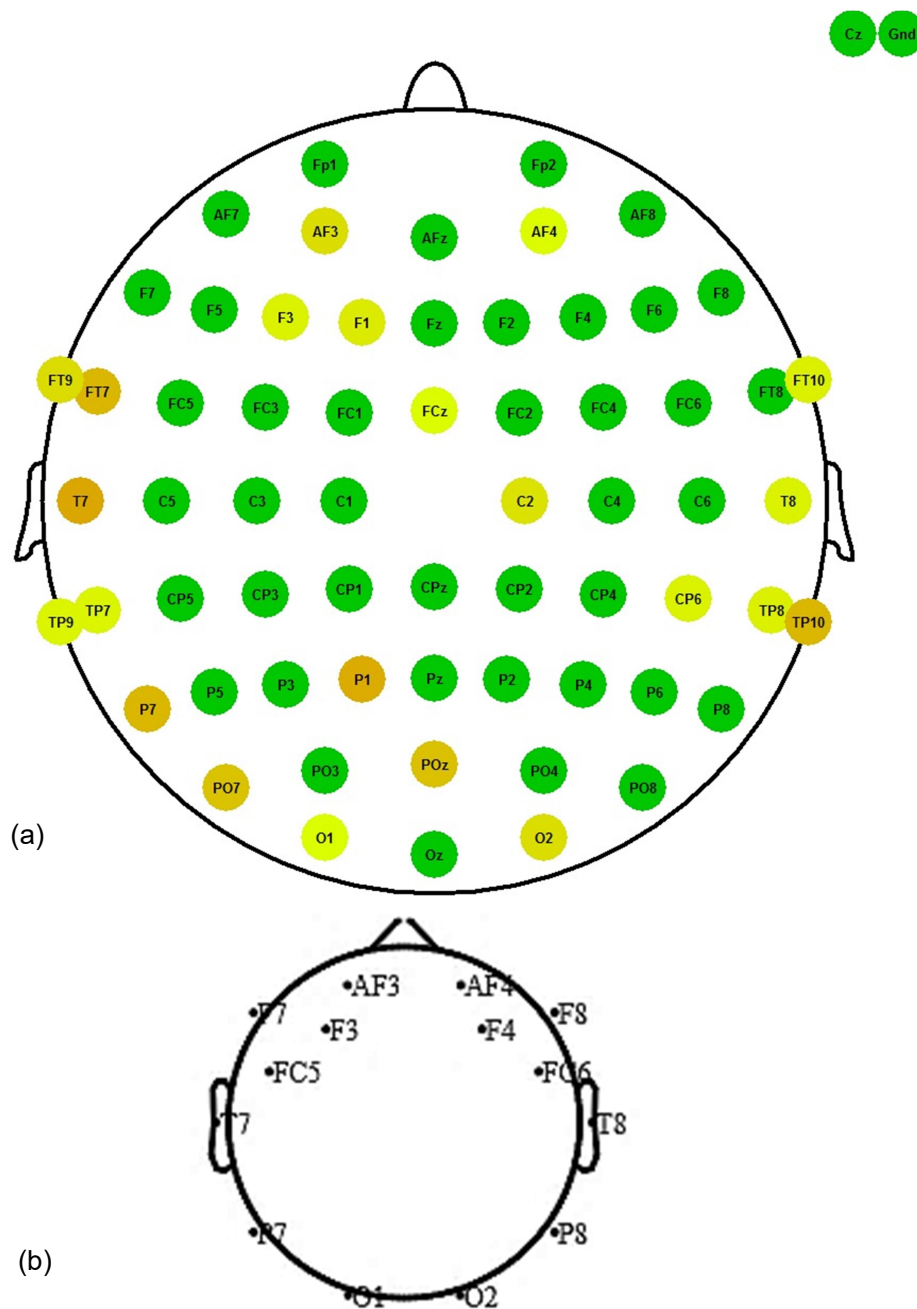
Electrodes were gelled in place using a blunt, sterilised needle and an electrolyte gel until electrode impedances were measured below 25 k $\Omega$ . The colour-coded impedance scale used is shown in Figure 6. Electrodes were initially shown as red in the recording software and on the electrodes themselves (through LEDs), and changed colour along the scale as gel was applied. This allowed the investigator to keep track of which electrodes had already been sufficiently gelled, as many electrodes had to be revisited and the gel swivelled with the needle to ensure proper contact.



**Figure 6: Brain Products impedance scale**

The Emotiv device's electrode contact was also shown in the relevant recording software, but only with four colours/conditions: grey indicated that the electrode was not properly connected to the device, red indicated that the electrode was not making contact with the subject's head, orange indicated that insufficient contact was made, and green indicated that the electrode contact was sufficient. Data was rejected if electrode contact was observed as any condition other than the green (sufficient contact) at any point in the recording.

Both the Emotiv and Brain Products devices make use of the 10-20 electrode placement system. The electrode locations for the 64 channels used with the Brain Products device are shown in Figure 11(a), while the electrode locations for the 14 channels used by the Emotiv device are shown in Figure 7(b). The electrodes shown for the Brain Products device are shown post-gelling, with the colours corresponding to the scale in Figure 6.



**Figure 7: Electrode configuration for (a) Brain Products and (b) Emotiv**

The subjects were asked to perform three tasks; the first of which was a seated, eyes closed recording to establish EEG signals during the relaxed state. Following this, the subjects performed a balance test and a Stroop test while still wearing the EEG device. All testing took place at the Stellenbosch Neuromechanics Lab at Coetzenberg.

### 3.2.2. Resting state procedure

The subject was asked to sit on a chair with his/her eyes closed. They rested their back against the backrest and remained as relaxed and still as possible. EEG readings were taken for 60 seconds.

### 3.2.3. Balance test procedure

This test required the subject to assume the tandem stance for 28 seconds with his/her eyes closed. A tandem stance is included in the SCAT analysis done by doctors for concussion screening, where twenty seconds of tandem stance is required. An additional eight seconds (40%) of recording time was included to provide for possible stumbles and trimming of the start and end of the recordings to minimise noise. EEG measurements were taken throughout the tandem stance task, which involves standing with one's feet in a line (one behind the other) with the non-dominant foot at the back. The participants were asked to keep their arms at their sides. The tandem stance is demonstrated in Figure 8. Participants were allowed to keep their shoes on or take them off, depending on how they felt most comfortable, but were asked to remain consistent with respect to this decision for all tandem stance tasks (i.e. both devices for healthy participants and for all sessions for the concussed participant).

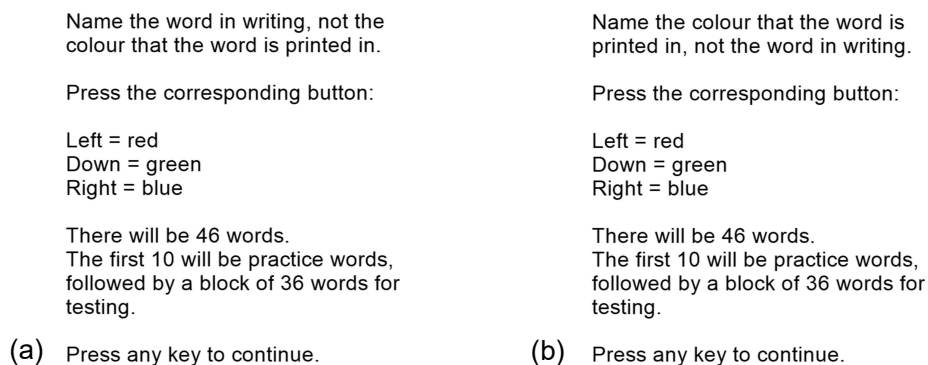


**Figure 8: Tandem stance**

### 3.2.4. Stroop test procedure

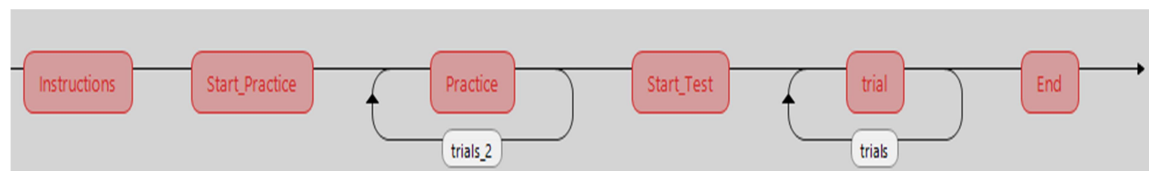
The Stroop test has already been introduced in Chapter 2, and involved the participant sitting in front of a computer screen. The names of colours appeared on the computer screen one at a time (with a white background), but the names were printed in a different colour ink to the meaning of the word. The colours used were red, blue and green. No word was printed in the colour that it represents, and was printed an equal number of times in each of the other colours. The words were ordered in such a way that no two consecutive words were written in the same colour or represent the name of the same colour.

The subject was given instructions on whether to select the word in writing (Stroop words test) or the colour that the word was printed in (Stroop colours test). He/she had to then select the corresponding button on a keyboard in front of them (left, right or down buttons) as quickly as possible. A test block of 36 words was preceded by a practice block of 10 words so that the subject could learn the colour-key associations. The responses of the test block were recorded, as well as the time taken to respond, and EEG recordings were taken throughout the test. The instruction screens presented to the participant for the Stroop tasks are given in Figure 9.



**Figure 9: Instruction screens for (a) Stroop words and (b) Stroop colours tasks**

At this point the participant was asked questions before starting the practice round, and was given another opportunity to ask questions after the practice round. The participant was asked to limit their movement to their fingers during the test block, and to avoid speaking unless it was an emergency. The flow diagram for a Stroop task is given in Figure 10. The Start\_Practice and Start\_Test blocks are pauses in the procedure to provide time for questions before proceeding.



**Figure 10: Flow diagram of Stroop task**

The words used in the Stroop tests are shown in Figure 11, read from left to right for each line from top to bottom. It should be noted that each line and column contain each word and colour an equal number of times, and that each word is printed an equal number of times in each of the other colours and never in the colour that it represents, as with the original Stroop study (Stroop, 1935) previously discussed in Chapter 2. This figure formed the basis of the computerised Stroop test, which was developed using Psychopy software (Peirce, 2002-2015).



**Figure 11: Stroop test with red, blue and green**

Half of the subjects performed the Stroop words task first, while the other half performed the Stroop colours task first. These groups were split evenly between the subjects who tested with the Emotiv and Brain Products devices first (e.g. roughly a quarter of the subjects performed the Stroop words first and tested with the Emotiv device first).

### 3.3. Data analysis

The data analysis was divided into an ERP analysis, a power spectrum analysis, and an analysis of the Stroop responses. This section will contain an overview of the methods followed for each analysis (Stroop task, resting state, tandem stance and response data). The filtering of the data, independent component analysis (ICA) and principal component analysis (PCA) will then be discussed in further detail, as well as the statistical analysis of the data. All EEG data was analysed using Matlab's EEGLab toolbox and response data was analysed using Microsoft Excel.

#### 3.3.1. ERPs and power spectra of Stroop tasks

Stroop task EEG data was filtered with a bandpass Finite Impulse Response (FIR) filter for 1 Hz to 40 Hz. The data was manually trimmed so that only data surrounding the test block was considered, and the data was epoched from -0.1 s to 0.6 s around the appearance of each stimulus (with the baseline removed based on the -0.1 s to 0 s interval). An ICA was run, and artifact components were pruned (rejected) for the concussed participant. Components were not pruned for the healthy participants, as this might influence the machine comparison. In comparing the Emotiv device to the Brain Products device, only steps which can be automated were included to prove that, if the Emotiv device is deemed sufficient, it can be used by non-experts in the field with minimal training. Since artifact component identification requires knowledge and training to perform, this was not included in the data from the machine comparison, thus removing the human element which might skew this aspect of the comparison.

Since the Brain Products device makes use of Cz as a reference for recordings and the Emotiv device makes use of the mastoids as a reference, all datasets of the healthy participants were re-referenced to a common average for comparison purposes. The Stroop task data of the concussed participant was also re-referenced to a common average for the purposes of comparison to the healthy control group, but the majority of analysis on the concussed participant's data was done using the Cz reference. In re-referencing, the ICA weights were also adjusted.

The common average reference method was chosen as the Emotiv device only contains electrodes in one of the hemispheres and using one of these electrodes as a reference may introduce a laterality bias into the data. It has also been found in research that the common average reference is best suited to P300 based applications when compared to eleven other referencing techniques (Alhaddad, 2012). Alhaddad found that the common average method performs better as the number of electrodes increased, but that it outperformed other methods (such as a right earlobe reference) with as few as eight electrodes. Re-referencing to a common average also improves the resolution for electrodes which lie close to a physical reference electrode such as Cz (Teplan, 2002). The common average re-referencing method was used by West and Bell (1997) to re-reference data collected from eight scalp locations. The data in West and Bell's study was collected while subjects were performing a Stroop task to investigate the effects of aging.

The epochs were averaged for each participant, resulting in ERP graphs, and the ERPs were compared and averaged for the two devices. PCAs were run for the data of the concussed participant and the spectrums plotted.

### **3.3.2. Resting state and tandem stance power spectra**

The resting state and tandem stance data was also bandpass filtered from 1 Hz to 40 Hz. The first and last second of data for each test was trimmed, such that all resting state data ran from 1 to 59 seconds, and all tandem stance data ran from 1 to 27 seconds. This eliminated any noise at the beginning or end of the task. An ICA was run for each dataset to identify and separate the components, followed by a PCA to recombine the weighted components into a single spectrum for the task. The artifact components were again pruned for the concussed participant, and the datasets of the healthy participants re-referenced to a common average. The spectrums were plotted for each participant, and maximum power values were identified for the delta (1 – 4 Hz), theta (4 – 8 Hz), alpha (8 – 13 Hz) and beta (13 – 30 Hz) frequency bands.

### **3.3.3. Responses**

The number of correct responses and response times per word were extracted for each Stroop task. These response measures were split between the participant's first and second Stroop words task, and between the participant's first and second Stroop colours task. The participants' response times were then averaged over the 36 responses for each of the four Stroop tests.

### **3.3.4. Filtering**

Data was first filtered using a highpass FIR filter with a cutoff frequency of 1 Hz, then filtered using a lowpass FIR filter with a cutoff frequency of 40 Hz. The filtering was done in two steps to effectively create a bandpass filter. The highpass cutoff frequency of 1 Hz was chosen as opposed to the frequently used 0.1 Hz cutoff frequency. A highpass cutoff frequency of 1 to 2 Hz was recommended by Winkler et al. (2015) if an ICA was to be run, as this improved the signal-to-noise ratio, single-trial classification accuracy and the percentage of near-dipolar ICA components (indicative of neurological components). The



lowpass filter cutoff frequency of 40 Hz was selected as this would eliminate any interference from power lines (usually around 50 Hz), and activity in the gamma frequency band (30 – 80 Hz) was not assessed.

### 3.3.5. Independent Component Analysis

The ICA algorithm is a statistical signal processing method, which separates the statistically independent source signals from signals which come from multiple channels (Ahirwal and londhe, 2012), and is useful in improving the detection of changes in EEG. The ICA method is outlined by Ahirwal and londhe (2012), and is included here.

If  $N$  is the number of EEG channels (electrodes), then there are  $N$  recorded signals. We name each recorded signal  $X_i$ , where  $i=1,\dots,N$ . We can then deduce that each signal is a linear mixture of  $N$  independent source signals ( $S_i$ ). We define  $X$  as the vector of recorded signals ( $X = [X_1, \dots, X_N]^T$ ),  $S$  as a vector of source signals ( $S = [S_1, \dots, S_N]^T$ ), and  $A$  as a mixing matrix (which is unknown), which gives us Equation 1.

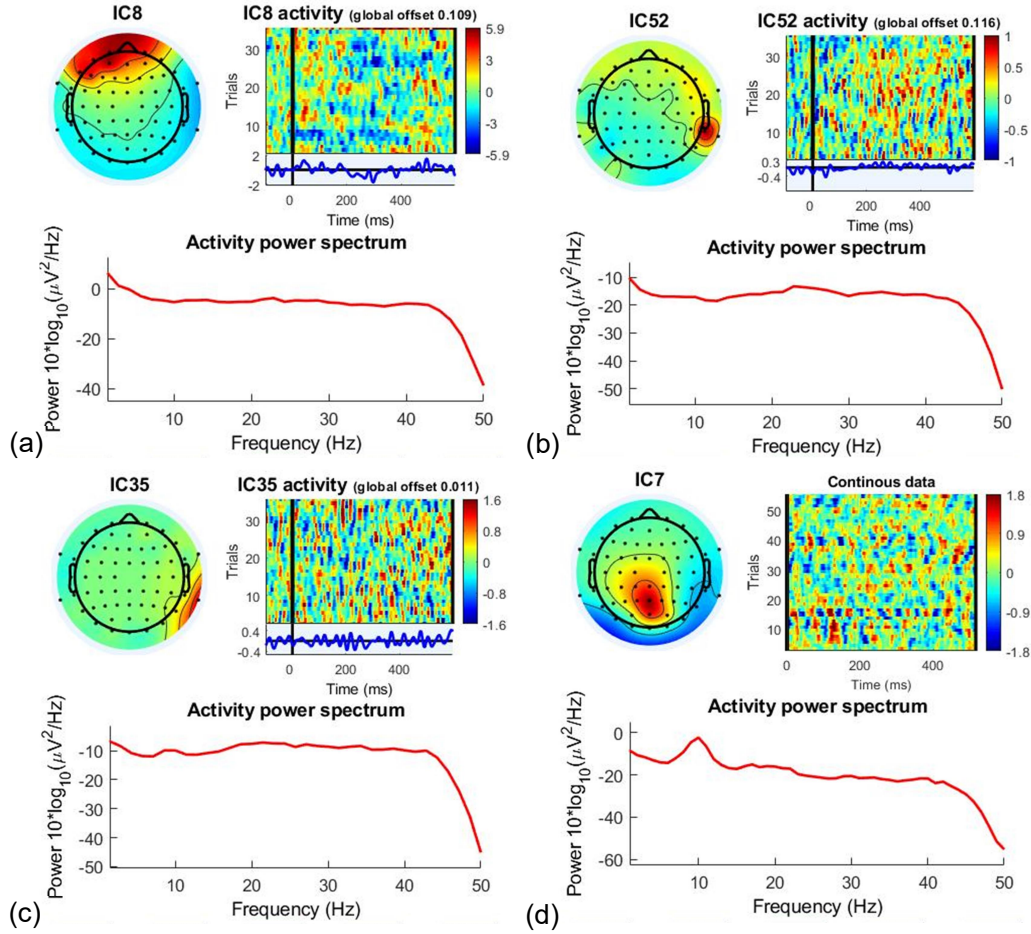
$$X = AS \quad (1)$$

In Equation 1,  $A$  and  $S$  are both unknown, thus  $S$  cannot be separated from  $X$  as traditional signal processing methods cannot solve the problem of blind source separation (Ahirwal and londhe, 2012). The separation matrix may be estimated using developed theories and algorithms (Ahirwal and londhe, 2015), resulting in the estimated separation matrix  $W$ . Equation 2 then holds, as the independent sources can be separated from the recorded signals using matrix  $W$ .

$$S = WX \quad (2)$$

The EEGLab ICA tool uses the logistic infomax ICA algorithm with the natural gradient feature or the extended ICA algorithm with sign estimation N training blocks (Ahirwal and londhe, 2012) to decompose the data. The extended ICA was used for this study as it better detects sources of activity such as line noise.

After the ICA was run, artifact components were rejected for the data of the concussed participant. Artifact components included components corresponding to muscle movement, eye movement and channel noise. An example of an artifact component relating to eye movement is shown in Figure 12(a), and is characterised by the far-frontal activity in the scalp map and a smooth activity spectrum. Figure 12(b) shows an example of an artifact component relating to channel noise, characterised by concentrated activity around a channel location in the scalp map, and an example of a muscle movement artifact is shown in Figure 12(c), characterised by activity in the scalp map which appears off the map and power at high frequencies (above 20 Hz) in the activity power spectrum. An example of a component relating to brain data is included in Figure 12(d), containing a discernable alpha peak, a dipole-like scalp map and regular activity across the trials (SCCN EEGLAB Tutorial, Data Analysis).



**Figure 12: Components of (a) eye movement, (b) channel noise, (c) muscle activity and (d) brain activity**

### 3.3.6. Principal Component Analysis

The PCA is a classical multivariate data analysis method, and is useful in feature extraction, data compression and dimensionality reduction (Ahirwal and londhe, 2012). It is also sometimes used for background noise reduction. It is a linear transformation, which takes components and transforms them while minimising the reconstruction error. The PCA procedure outlined here was given by Ahirwal and londhe (2012) in an article on power spectrum analysis. This linear transformation is described by Equation 3, where the row vectors of  $B$  correspond to the normalized orthogonal eigenvectors of the data covariance matrix.

$$C = B_u \quad (3)$$

Singular value decomposition is considered a simple approach to the PCA (Ahirwal and londhe, 2012). The singular value decomposition of the covariance matrix  $R_u$  then results in Equation 4. Matrix  $U_u$  is the eigenvector matrix and  $D_u$  is



the diagonal matrix with the eigenvalues of  $R_u$  on its diagonal. We the set  $B = U'_u$  for the PCA.

$$R_u = U_u D_u U'_u \quad (4)$$

All components generated by the ICA (except the pruned components for the concussed participant) are reduced to one component using the PCA (Matlab's PCA function was used). This gives more accurate readings of the power spectrum associated with different frequency bands, particularly since the PCA takes into account the weights of the components generated by the ICA.

### 3.3.7. Statistical analysis

A statistical analysis was run on the results of the healthy participants. The ERP peaks for the Stroop colours and Stroop words tasks, the power spectrum peaks for the resting state and tandem stance data, the number of correct responses for the Stroop tasks and the average response time per Stroop task were analysed using paired observation t-tests, Pearson correlation coefficients and statistical power. For the statistical analysis it was assumed that the data was approximately normally represented.

The initial null hypothesis for each t-test was that there is no difference between the machines, and that the difference between the means of the two samples is thus zero. The alternate hypothesis was that there is a difference between the means of the samples. A two-tailed t-test was conducted, thus the null and alternate hypotheses are given by Equations 5 and 6 respectively, where  $\mu_d$  is the mean of the differences.

$$H_0 : \mu_d = 0 \quad (5)$$

$$H_1 : \mu_d \neq 0 \quad (6)$$

The test statistic was given by Equation 7, where  $\bar{d}$  is the sample mean of the differences,  $\mu_0$  is the hypothesised mean difference,  $\sigma_d$  is the standard deviation of the differences and  $n$  is the sample size.

$$t = \frac{\bar{d} - \mu_0}{\sigma_d / \sqrt{n}} \quad (7)$$

This test statistic was used to determine a  $P$  value, which measures how compatible the data is with the null hypothesis (i.e. the likelihood of obtaining the sample data if the null hypothesis is true). A sufficiently low  $P$  provides enough evidence that the null hypothesis can be rejected. For the purposes of this study, null hypotheses were rejected for  $P$  values less than 0.1 (i.e. there is less than a 10% chance of obtaining the sample in question if the null hypothesis is true).

Pearson correlation coefficients were used to determine whether there was a linear relation between the datasets. If the results obtained from the two machines were the same, or differed by a scaling factor, the Pearson correlation coefficient would return a value close to one. If the data correlated negatively, a value close to negative one would be obtained, while a value of zero indicated that there is no linear correlation between the datasets.

The statistical power for each test was determined using the Matlab function *tpower*, written by Jason Augustyn (2005). The power calculated by this function required inputs of the sample size, the chosen value of alpha (0.1 in this case) and the  $d$  value for the comparison, calculated using Equation 8. In Equation 8,  $\mu_1$  is the mean of observation 1,  $\mu_2$  is the mean of observation 2,  $\sigma_1$  is the standard deviation of dataset 1,  $\sigma_2$  is the standard deviation of dataset 2 and  $PCC$  is Pearson's correlation coefficient.

$$d = \frac{|\mu_1 - \mu_2|}{\sqrt{\sigma_1^2 + \sigma_2^2 - 2(\sigma_1)(\sigma_2)(PCC)}} \quad (8)$$

For the purposes of this study, the threshold for sufficient statistical power was set at 90%, as recommended by Dumas-Mallet et al. (2017).

Statistical analyses were run on the ERP peak data to include and exclude outliers, as well as to hypothesize a mean difference of zero and a mean difference equal to the difference in overall averaged peak amplitudes in the ERP graphs in order to determine whether the ERP graphs obtained are an accurate depiction of the individual results. ERP peak values were identified using the average ERP graphs as a guideline. For the P300 peaks of the Brain Products device, a latency of 250 ms was observed for the average graphs, thus the maximum peak between 200 and 300 ms was identified for each of the individual graphs. Similarly, a latency of 400 ms was observed for the P300 peaks of the Emotiv device, thus individual peaks were identified between 350 and 450 ms for the individual graphs. For the N200 peaks, maximum peaks were identified between 150 and 250 ms for the Brain Products data and between 350 and 450 ms for the Emotiv data.

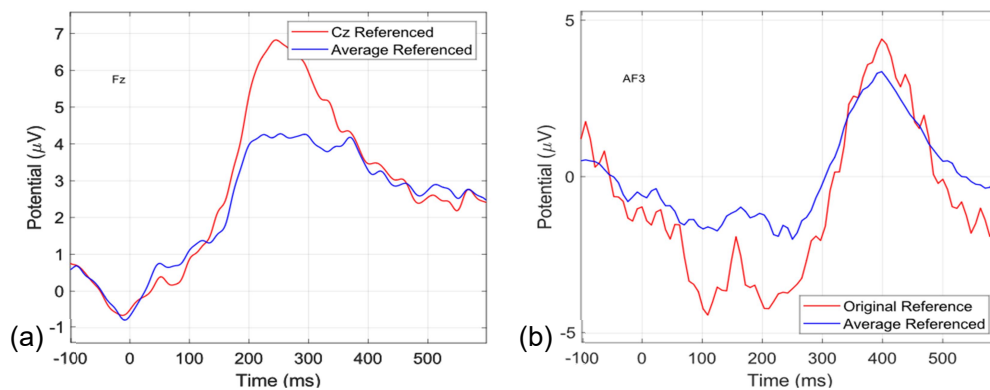
Statistical analyses were run on the power spectrum peak data, as well as on the peak data with the baselines removed to determine whether the trends between peaks obtained by the devices were comparable. Lastly a statistical analysis was run on the response data to determine whether practice effects played a significant role.

## 4. Healthy participants: Results and preliminary discussion

This chapter includes the results and discussion of the testing of the 22 healthy participants. First, the effect of re-referencing the data to a common average is included, along with the effects of changing the sampling rate of the Brain Products data. Next, an overall comparison of the two machines is included, as well as the effects of the order of testing (with respect to devices). Finally, the practice effects relating to the Stroop tasks are investigated.

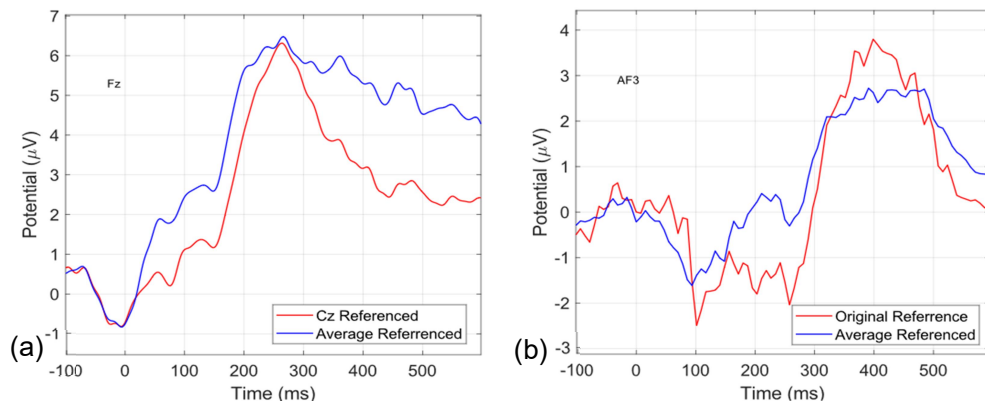
### 4.1. Re-referencing and sampling rates

As mentioned in Chapter 3, the data from the two machines had to be re-referenced to the common average method for comparison purposes. Figure 13(a) shows the effects on ERPs of converting the Brain Products data from the Stroop words test to a common average, while Figure 13(b) shows the effects of converting the Emotiv data from the Stroop words test.



**Figure 13: Re-referencing the (a) Brain Products (shown for Fz) and (b) Emotiv (shown for AF3) Stroop words data**

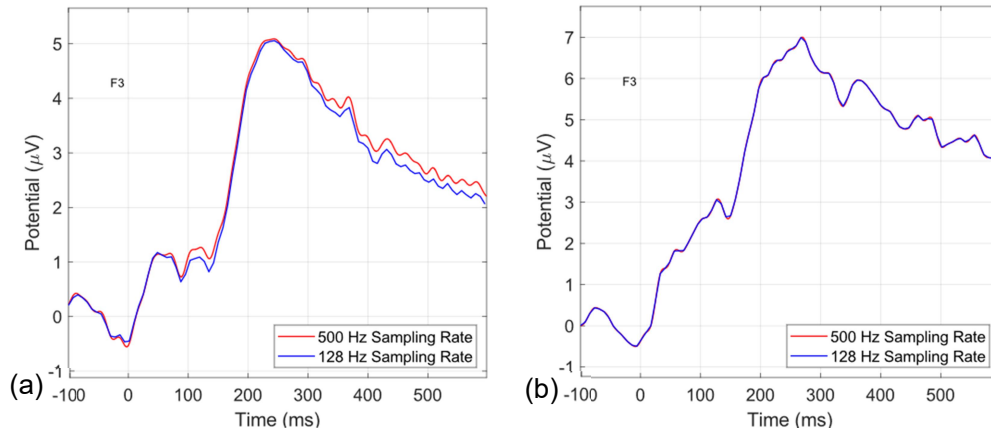
Similarly, Figure 14 shows the results of converting the Brain Products data and Emotiv data for the Stroop colours test to a common average.



**Figure 14: Re-referencing the (a) Brain Products (shown for Fz) and Emotiv (shown for AF3) Stroop colours data**

It is clear from Figure 13 and Figure 14 that re-referencing the data did not affect the latency of the P300 peaks. However, the amplitudes of the peaks were slightly altered. This change in amplitude can be expected, as the amplitude is dependent on the distance of the measuring electrode from the reference point. Since the reference has been changed to a common average (for each test), the amplitudes are expected to change by varying degrees.

In changing the sampling rate of the Brain Products data from 500 Hz to match the sampling rate of the Emotiv (128 Hz), the effects shown in Figure 15 for the Stroop words and Stroop colours were obtained.

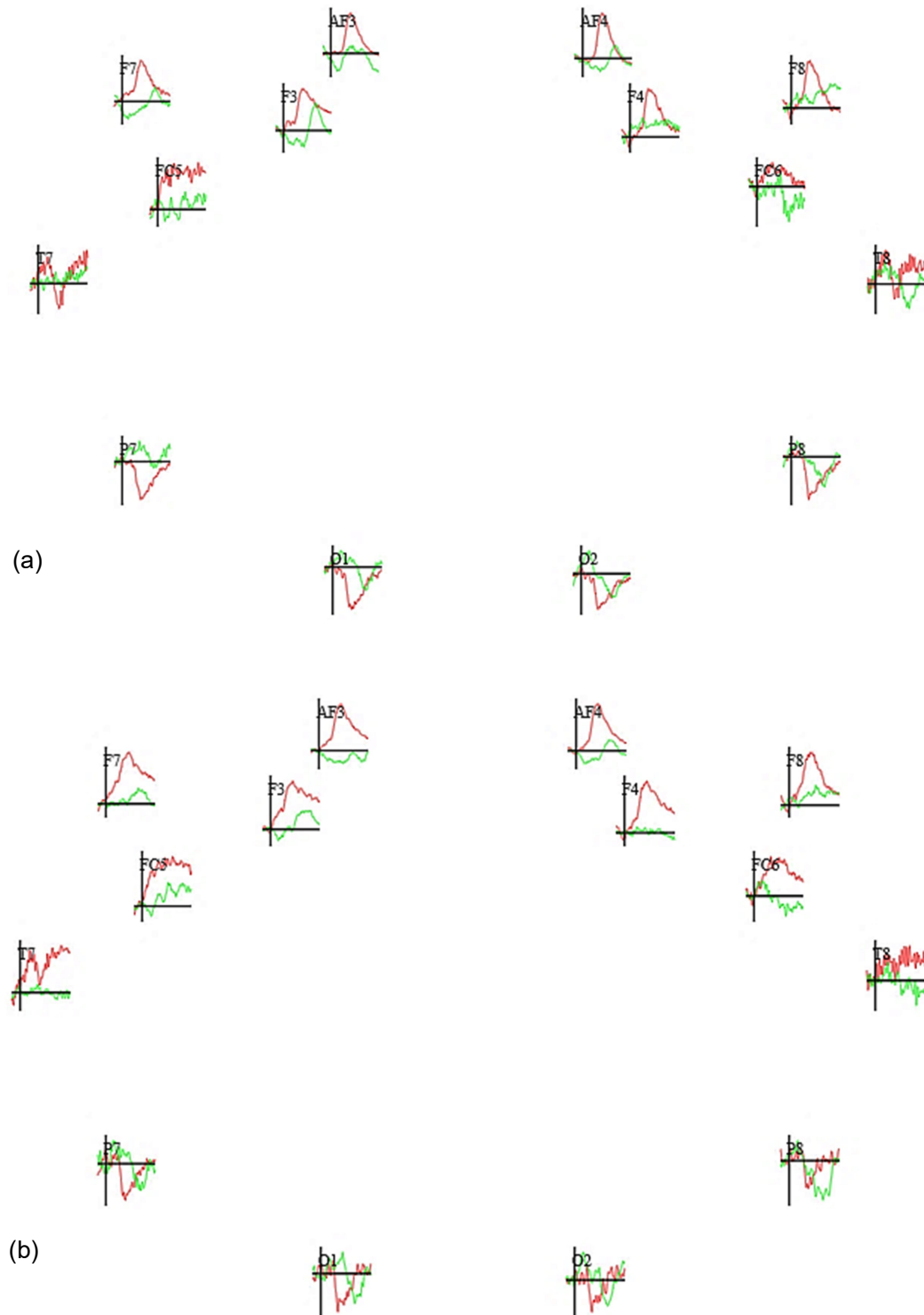


**Figure 15: Downsampling the Brain Products (a) Stroop words and (b) Stroop colours data (shown for F3)**

It is clear from Figure 15 that changing the sampling rate did not influence the amplitude or latency of the P300 peak. The downsampling will therefore not influence the machine comparison.

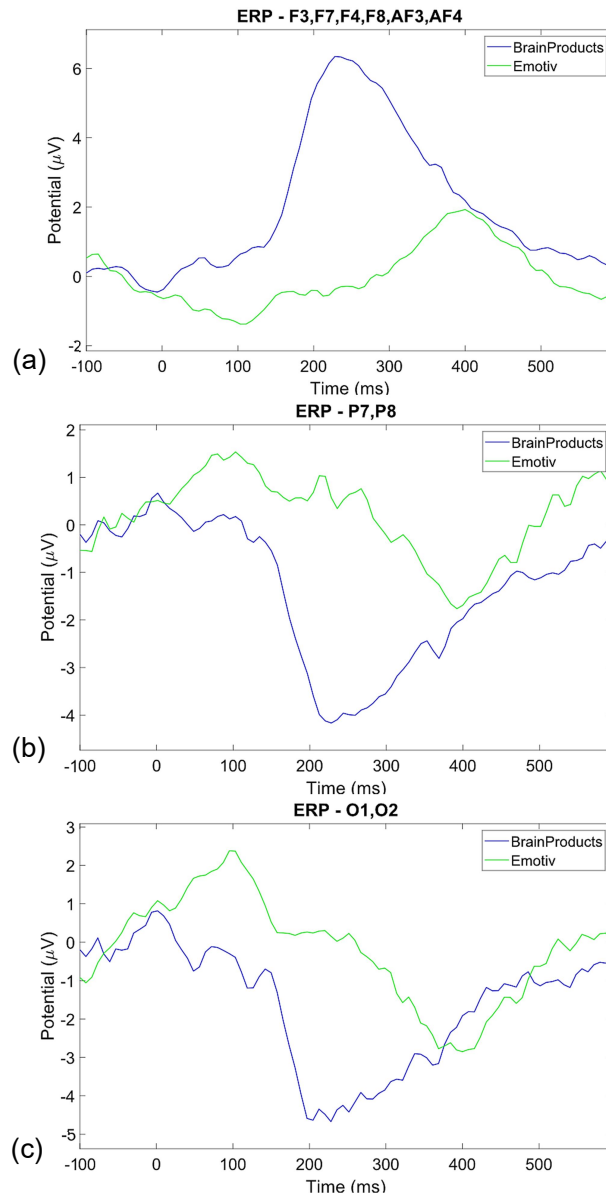
## 4.2. Overall comparison

The Brain Products ERP data (after re-referencing and downsampling) was compared to the Emotiv ERP data (after re-referencing). Figure 16 shows the ERP comparisons for the channels that the two devices have in common plotted in the scalp locations of the channels. The Brain Products ERPs are shown in red, while the Emotiv ERPs are shown in green. It is important to note that this figure is included to provide an overview of the responses, but that no other information is expected to be gained from this figure. More detailed graphs of the responses are included in subsequent figures. The overview of the ERPs in Figure 16 shows positive peaks in the anterior region and negative peaks in the posterior region. It can already be noticed that similar trends are presented for the Brain Products and Emotiv data, with differences in amplitude and latency of these trends evident.

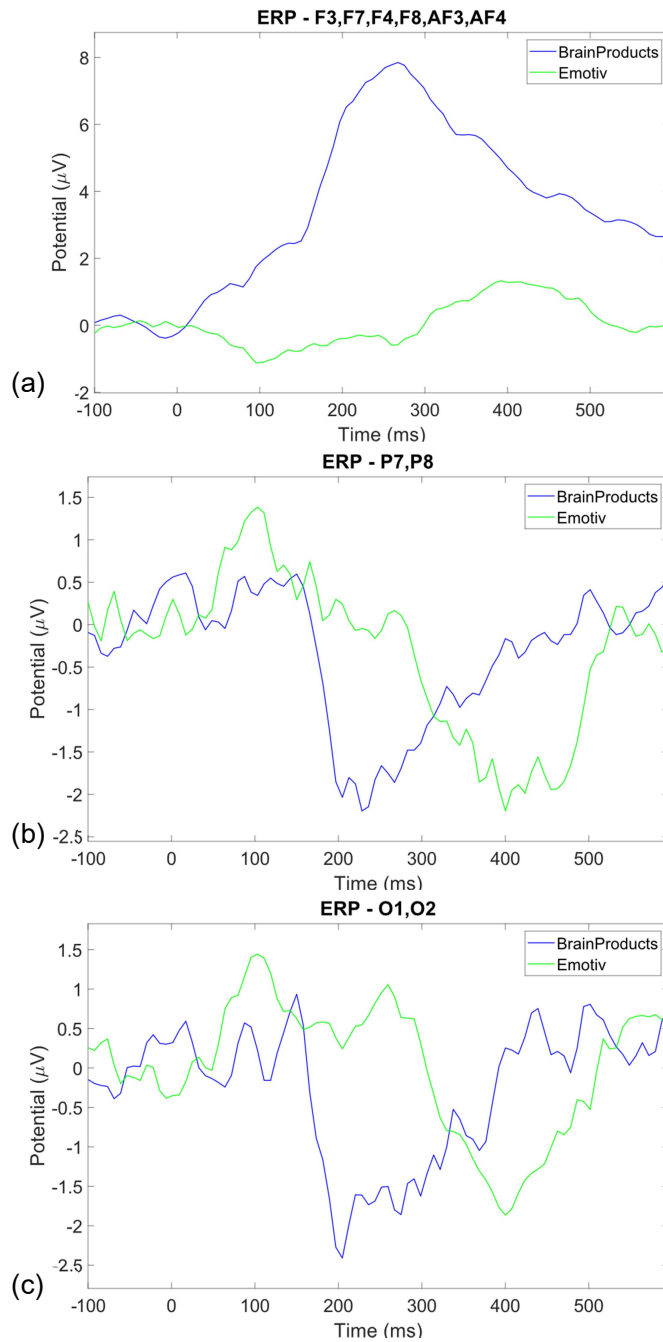


**Figure 16: Brain Products (red) and Emotiv (green) (a) Stroop words and (b) Stroop colours ERP comparison**

Since previous Stroop studies (discussed in Section 2.6) considered the Fz, Pz, Cz and Oz electrodes (all along the middle line of the scalp), and the Emotiv has no electrodes in these locations, the frontal (F3, F4, F7, F8, AF3 and AF4) channels were averaged, as well as the parietal (P7 and P8) and occipital (O1 and O2) channels. This should provide information to compare the ERPs to the Fz, Pz and Oz electrode ERPs obtained in literature, since it is clear from Figure 16 that the ERPs in these regions follow similar trends. The resulting ERPs for the Stroop words test are shown in Figure 17, and the resulting ERPs for the Stroop colours test are shown in Figure 18.



**Figure 17: Stroop words ERP comparison for (a) Frontal, (b) Parietal and (c) Occipital regions**



**Figure 18: Stroop colours ERP comparison for (a) Frontal, (b) Parietal and (c) Occipital regions**

From previous literature, a P300 peak is expected in the frontal region, and an N200 peak is expected in the parietal and occipital regions (Broglia et al., 2009). The P300 peak is observed in the frontal region at a latency of about 250 ms for the Brain Products device and about 400 ms for the Emotiv device for both Stroop tasks, and has a reduced amplitude for the Emotiv device when compared



to the Brain Products device. Despite this, the frontal region ERPs for the two devices seem to follow similar trends, especially for the Stroop words task. The N200 peaks were observed with a latency of 200 ms for the Brain Products device, with very similar trends occurring at 400 ms for the Emotiv device (with a larger amplitude difference for the Stroop words parietal region ERP). Table 1 contains a summary of the results of the statistical analysis conducted on the amplitudes of the ERP peaks of the Stroop words and Stroop colours tasks. Details of the individual peaks are included in Appendix D.

**Table 1: Statistical analysis of overall ERPs**

Activity		Hypothesised mean difference [µV]	Pearson Correlation Coefficient	P(T<=t)	Power	Reject H <sub>0</sub> ? (P<0.1)	Strong statistical power? (>0.8)
Stroop words ERPs	F	0	0.707	0.0548	0.622	Yes	No
	P	0	0.760	0.111	0.477	No	No
	O	0	0.697	0.208	0.338	No	No
Stroop colours ERPs	F	0	0.479	0.0166	0.807	Yes	Yes
	P	0	0.641	0.793	0.0803	No	No
	O	0	0.548	0.778	0.0830	No	No

Key:

F = Frontal region

P = Parietal region

O = Occipital region

From Table 1, we can observe that only the ERPs from the frontal region in the Stroop colours test have sufficient statistical power to draw conclusions. We therefore conclude from the statistical analysis that there are significant differences between the machines with respect to the frontal region Stroop colours ERPs. The results of the Stroop words test and other regions of the Stroop colours test remain inconclusive due to insufficient statistical power. The Pearson correlation coefficients indicate moderate (0.5 to 0.7) to strong (0.7 to 1) positive linear relationships between the ERP peaks from the two devices. This may indicate that the ERPs from the two machines differ by a scaling factor, but stronger statistical power and further investigation would be required to confidently draw these conclusions.

Two subjects were identified as outliers as a result of unusually high ERP peaks for the Stroop words test (for both devices) and two were identified as outliers for the Stroop colours test. The statistical analysis was repeated with these outliers removed, yielding the results summarised in Table 2.

**Table 2: Statistical analysis of overall ERPs with outliers removed**

Activity		Hypothesised mean difference [ $\mu$ V]	Pearson Correlation Coefficient	P(T $\leq$ t)	Power	Reject $H_0$ ? (P<0.1)	Strong statistical power? (>0.8)
Stroop words ERPs	F	0	0.012	0.100	0.499	No	No
	P	0	0.134	0.462	0.169	No	No
	O	0	-0.100	0.710	0.096	No	No
Stroop colours ERPs	F	0	0.356	2.42E-05	0.999	Yes	Yes
	P	0	-0.121	0.793	0.401	No	No
	O	0	0.218	0.157	0.305	No	No

Key:

F = Frontal region

P = Parietal region

O = Occipital region

Removing the outliers decreased the statistical power of the Stroop words ERP data and increased the statistical power of the Stroop colours ERP data. The  $P$  value for the Stroop words frontal ERPs increased to slightly above the rejection threshold, but without sufficient statistical power this does not change any of the conclusions drawn from Table 1. Interestingly, the removal of the outliers affected the Pearson correlation coefficients to a large extent. They no longer indicate linear relationships, but higher statistical power is again necessary to draw conclusions.

An investigation into whether the ERP graphs given in Figure 17 and Figure 18 describe a statistically significant difference in amplitude yielded results, which are given in Table 3. The outliers were included for this analysis since they were included in the graphs.

**Table 3: Statistical analysis of overall ERP graphs**

Activity		Hypothesised mean difference [ $\mu$ V]	Pearson Correlation Coefficient	P(T $\leq$ t)	Power	Reject $H_0$ ? (P<0.1)	Strong statistical power? (>0.8)
Stroop words ERPs	F	4.41	0.707	0.792	0.622	No	No
	P	2.41	0.760	0.00343	0.477	Yes	No
	O	1.82	0.697	0.0231	0.338	Yes	No
Stroop colours ERPs	F	6.52	0.479	0.748	0.807	No	Yes
	P	0.001	0.641	0.793	0.0803	No	No
	O	0.545	0.548	0.599	0.0830	No	No

Key:

F = Frontal region

P = Parietal region

O = Occipital region

It can be observed from the results in Table 3 that the  $P$  values of the frontal region Stroop words and Stroop colours peaks increased greatly to a point which strongly supports the null hypothesis for both tests. However, while the Stroop words statistical power can be considered almost sufficient, the power for neither test is at the 80% limit where conclusions can be drawn.

While the graphs in Figure 17 and Figure 18 do show similar trends for the ERPs obtained with the two devices, statistics have shown that the high variability of the individual ERPs means that the devices cannot reliably measure a constant amplitude difference equal to the difference indicated by the peaks. The differing latencies of the individual peaks have resulted in overall peaks which do not describe a consistent mean difference across the peaks, indicating high variability in latencies. However, these tests should be repeated with more participants to ensure sufficient statistical power for all variables.

The results of the statistical analysis on the power spectrum data are given in Table 4. From this table, it can be concluded with sufficient statistical power that the delta, theta and beta peaks for the resting state power spectra of the two machines differ, as well as the delta peaks for the tandem stance power. No other results hold sufficient statistical power to draw conclusions, but it should be noted that the rejection of the null hypothesis for the tandem stance theta power has close to sufficient statistical power. The Pearson correlation coefficients show no linear correlations between the peak powers.

**Table 4: Statistical analysis of power spectrum results**

Activity		Hypothesised mean difference [ $\mu V$ ]	Pearson Correlation Coefficient	$P(T \leq t)$	Power	Reject $H_0$ ? ( $P < 0.1$ )	Strong statistical power? ( $> 0.8$ )
Resting state	Delta	0	0.257	3.51E-05	0.999	Yes	Yes
	Theta	0	0.257	0.00130	0.995	Yes	Yes
	Alpha	0	0.0387	0.226	0.257	No	No
	Beta	0	0.0711	0.0194	0.885	Yes	Yes
Tandem	Delta	0	0.219	0.00610	0.933	Yes	Yes
	Theta	0	0.121	0.0376	0.745	Yes	No
	Alpha	0	0.214	0.863	0.0697	No	No
	Beta	0	-0.109	0.530	0.145	No	No

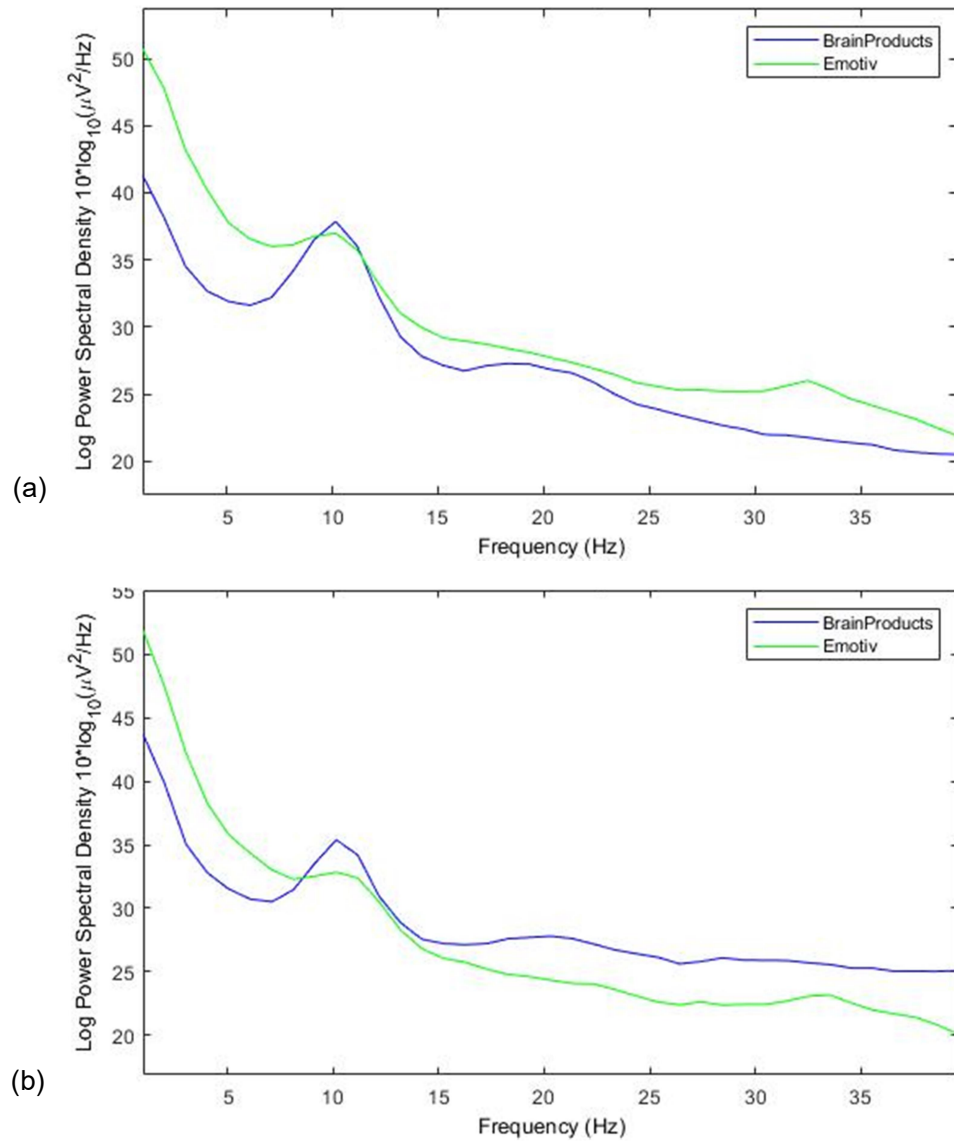
In order to eliminate a potential baseline difference from skewing the results, the mean of the power spectrum for each dataset was subtracted and the statistical analysis rerun. The results of this are shown in Table 5. From this analysis, it can be concluded that the null hypothesis can be rejected with sufficient statistical significance for the resting state delta, alpha and beta frequency bands (as well as close to sufficient statistical power for the theta band). Additionally, the null hypothesis can be rejected for the tandem stance delta, alpha and beta

frequency bands. The Pearson correlation coefficients again show no linear correlations between the peaks powers.

**Table 5: Statistical analysis of power spectrum results with mean removed**

Activity		Hypothesised mean difference [µV]	Pearson Correlation Coefficient	P(T≤t)	Power	Reject H <sub>0</sub> ? (P<0.1)	Strong statistical power? (>0.8)
Resting state (mean removed)	Delta	0	0.0394	0.00172	0.962	Yes	Yes
	Theta	0	0.132	0.0261	0.746	Yes	No
	Alpha	0	0.261	0.000190	0.995	Yes	Yes
	Beta	0	0.326	0.0123	0.840	Yes	Yes
Tandem (mean removed)	Delta	0	0.284	0.0129	0.851	Yes	Yes
	Theta	0	0.320	0.180	0.416	No	No
	Alpha	0	0.329	0.0128	0.871	Yes	Yes
	Beta	0	0.242	0.0177	0.807	Yes	Yes

The results of the power spectrum analysis seem to indicate that the power spectrums of the two machines are significantly different. However, Figure 19 shows the comparisons of the average power spectrums graphically for the resting state and tandem data. Since the power spectrums of both devices seem indicative of brain activity with distinguishable alpha peaks, there remains the possibility that the power spectrums obtained with the Emotiv device may be sensitive enough to detect the subtle deficits caused by concussion.

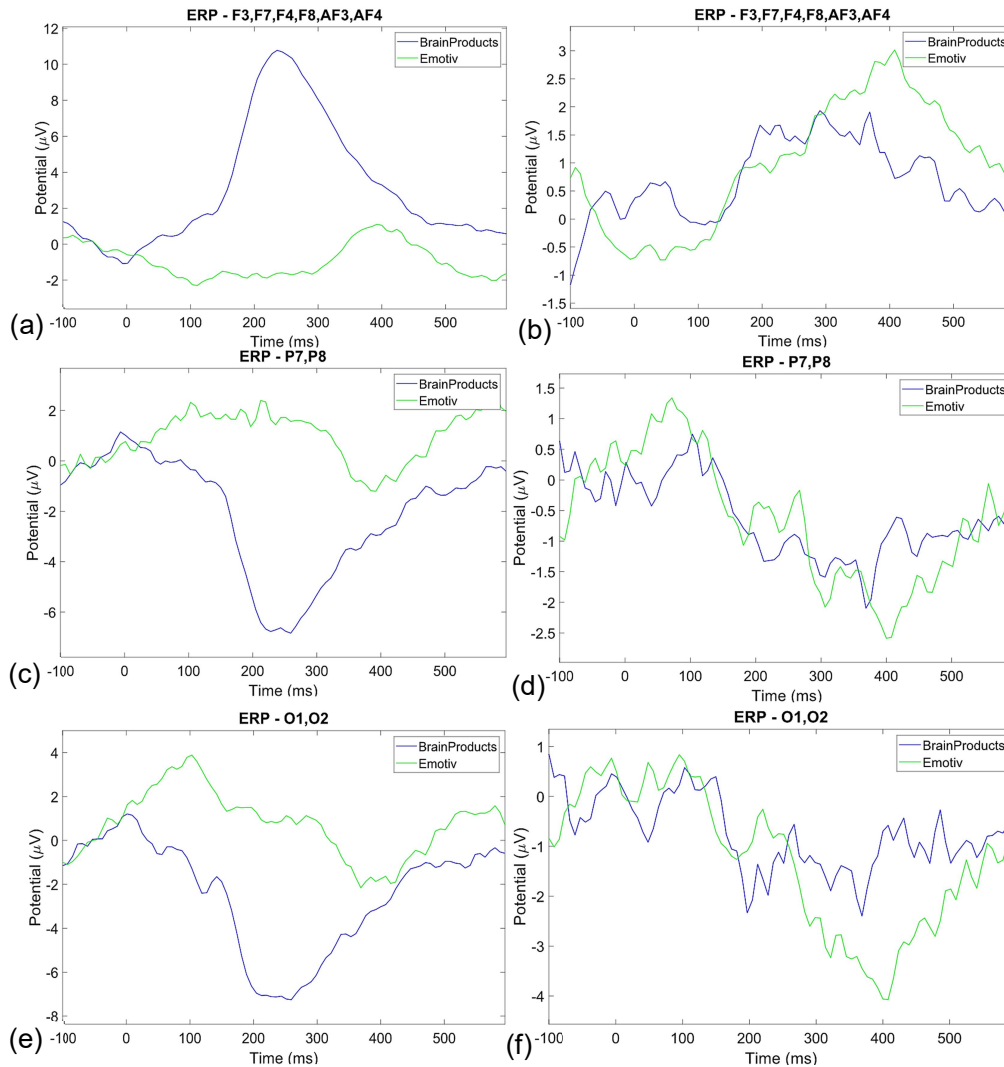


**Figure 19: Power spectrum comparison of (a) the resting state data and (b) the tandem stance data**

### 4.3. Machine order effects

Since ten participants conducted the testing with the Brain Products device first and twelve participants conducted the testing with the Emotiv device first, the order of machines for testing may have had an influence on the data. This section contains the results and discussion of an investigation into those effects.

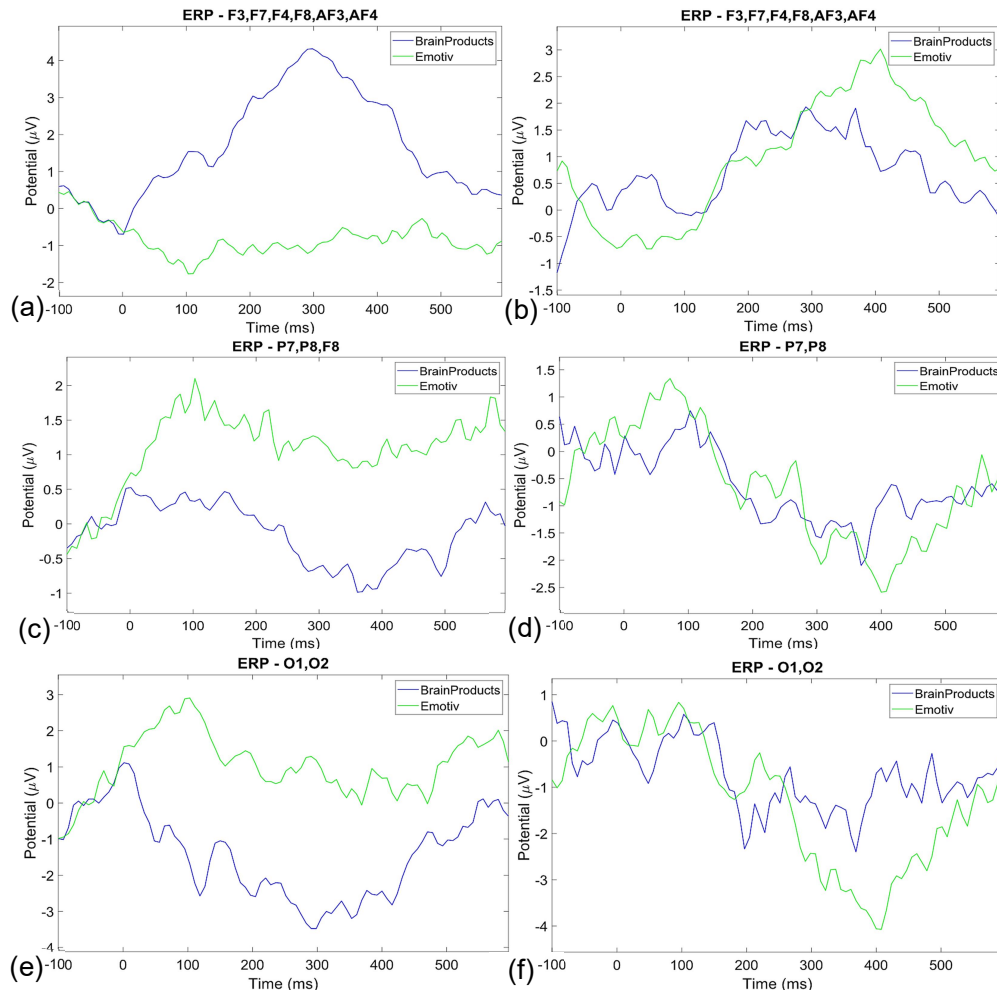
Figure 20 contains Stroop words test ERP comparisons between the two devices for the subjects who tested with the Emotiv device first and subjects who tested with the Brain Products device first.



**Figure 20: Machine order effects on Stroop words ERPs for frontal (a) Emotiv first and (b) Brain Products first, parietal (c) Emotiv first and (d) Brain Products first and occipital (e) Emotiv first and (f) Brain Products first regions**

Large differences were observed between the average ERPs of the subjects who tested with the Emotiv device first and subjects who tested with the Brain Products device first. Some differences were expected in the Emotiv data due to residual gel in the subjects' hair after testing with the Brain Products device first, but this does not account for the differences in the Brain Products data between the two tests. Differences could be accounted to practice effects, but further investigation is warranted.

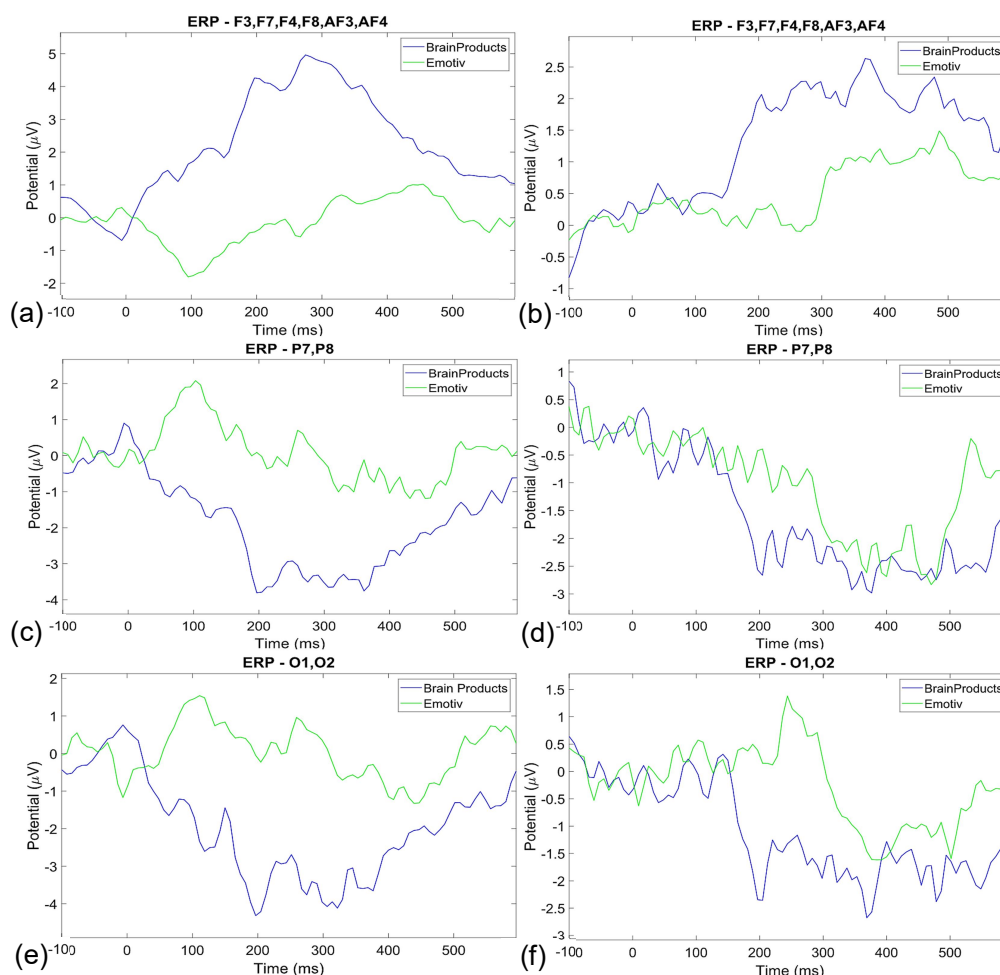
Investigations into the split of the group revealed that both outlier participants identified previously tested with the Emotiv device first. After removing the ERPs of these participants, the ERPs in Figure 21 were obtained for the participants who tested with the Emotiv device first. The ERPs of the participants who tested with the Brain Products device first are included again for the sake of comparison.



**Figure 21: Machine order effects on Stroop words ERPs for frontal (a) Emotiv first and (b) Brain Products first, parietal (c) Emotiv first and (d) Brain Products first and occipital (e) Emotiv first and (f) Brain Products first regions with outliers removed**

The ERPs seem to be more consistent with the outliers removed. The ERPs of the Brain Products data have similar amplitudes and trends for the two conditions, while the ERPs of the Emotiv data have more defined peaks with larger amplitudes in the tests where the subjects performed testing with the Brain Products device first (i.e. larger amplitudes in the second Stroop words test). As mentioned, this could be as a result of practice effects, which are investigated in the next section. This idea is supported by the larger amplitudes of the peaks of the Brain Products data in the ERPs of the subjects who performed testing with the Emotiv device first for the frontal and occipital regions.

A similar investigation into the ERPs of the Stroop colours test was conducted. The data from the two outliers was again removed for a more fair comparison, and the ERPs in Figure 22 were obtained. The ERPs are again divided between participants who tested with the Emotiv device first and those who tested with the Brain Products device first.



**Figure 22: Machine order effects on Stroop colours ERPs for frontal (a) Emotiv first and (b) Brain Products first, parietal (c) Emotiv first and (d) Brain Products first and occipital (e) Emotiv first and (f) Brain Products first regions with outliers removed**



Again, similar trends are observed for the Brain Products data for the two conditions, with slightly larger amplitudes in the peaks of the ERPs obtained from the participants who tested with the Emotiv device first. The Emotiv data also showed more defined peaks in the ERPs obtained from the subjects who tested with the Brain Products device first. This supports the findings of the Stroop words investigation that practice effects may have played a role. The residual gel in the participants' hair that tested with the Brain Products device first may also account to some extent for the more distinct Emotiv ERP peaks obtained for those participants.

#### 4.4. Practice effects

This section contains the results of an investigation into the practice effects associated with performing the Stroop tests twice. The numbers of correct responses to the Stroop tests, as well as the response times, were recorded. The practice effects on the ERPs were also investigated and the results included in this section.

The average number of correct responses and average response time for each test are given in Table 6. A statistical analysis was run on this data and the results are included in

Table 7. The results of individual participants are included in Appendix F.

**Table 6: Average response times (per word/colour)**

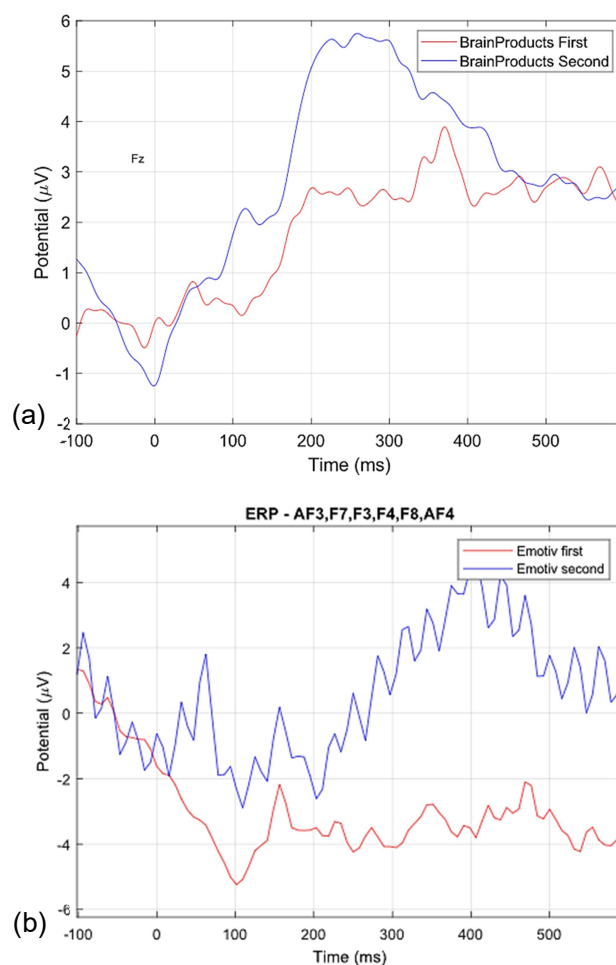
Test	Task	Average time per word/colour [s]	Average number of correct responses (out of 36)
Stroop words	First	0,866	35.36
	Second	0,835	35.5
Stroop colours	First	0,980	35.36
	Second	0,915	35.5

**Table 7: Statistical analysis of practice effects**

Activity		Hypothesised mean difference [µV]	Pearson Correlation Coefficient	P(T<=t)	Power	Reject H <sub>0</sub> ? (P<0.1)	Strong statistical power? (>0.8)
Response times	Words (first vs. second)	0	0.646	0.458	0.171	No	No
	Colours (first vs. second)	0	0.758	0.140	0.416	No	No
Number of correct responses	Words (first vs. second)	0	0.420	0.480	0.410	No	No
	Colours (first vs. second)	0	0.210	0.544	0.158	No	No

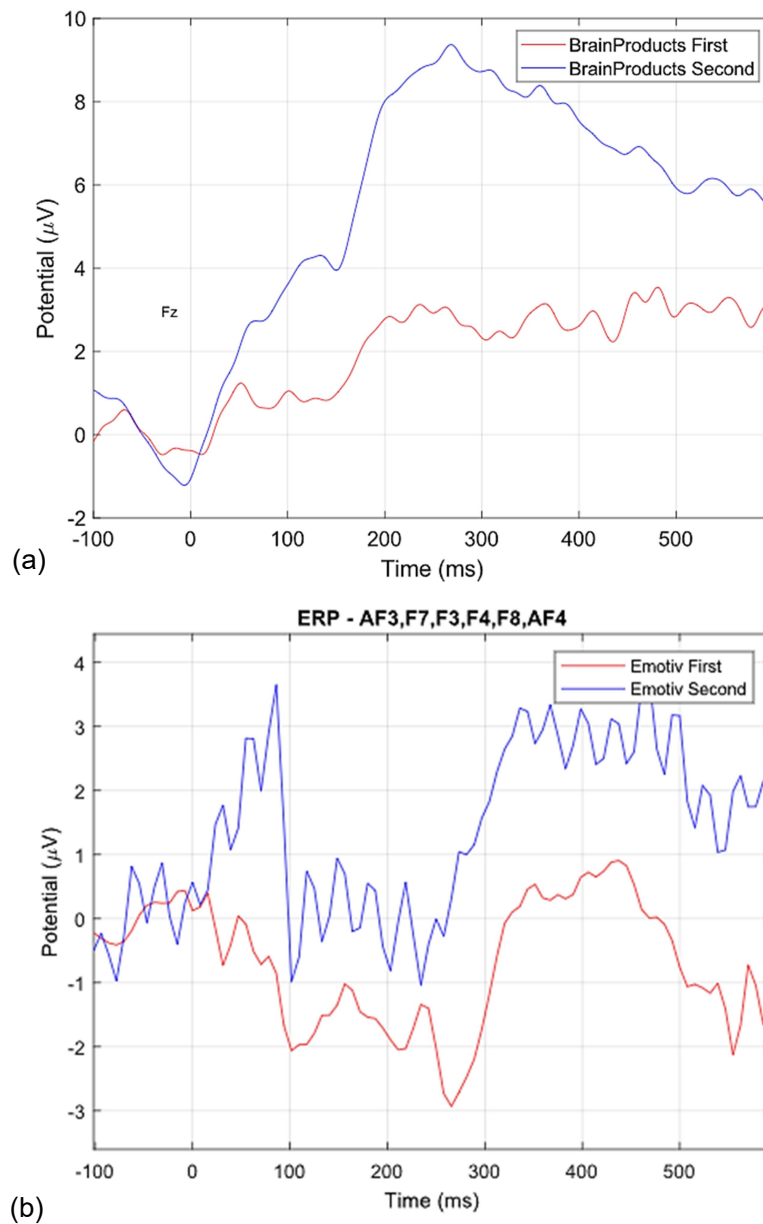
The statistical analysis indicated that there are no statistically significant differences between the first and second Stroop tasks. However, since statistical power was low, this may not be the case after further investigation. Moderate to high Pearson correlation coefficients for some of the tasks may indicate linear relationships between the response data (which can be expected if the responses are the same for the two tasks), but should also be further investigated.

Figure 23 shows the Stroop words ERPs for the Brain Products and Emotiv devices split into the two groups that did the tests with the relevant device first (i.e. it is the subjects' first Stroop words test) and second (i.e. it is the subject's second Stroop words test). These ERPs were obtained using the original device references and sampling rates, as the devices were not being compared to each other.



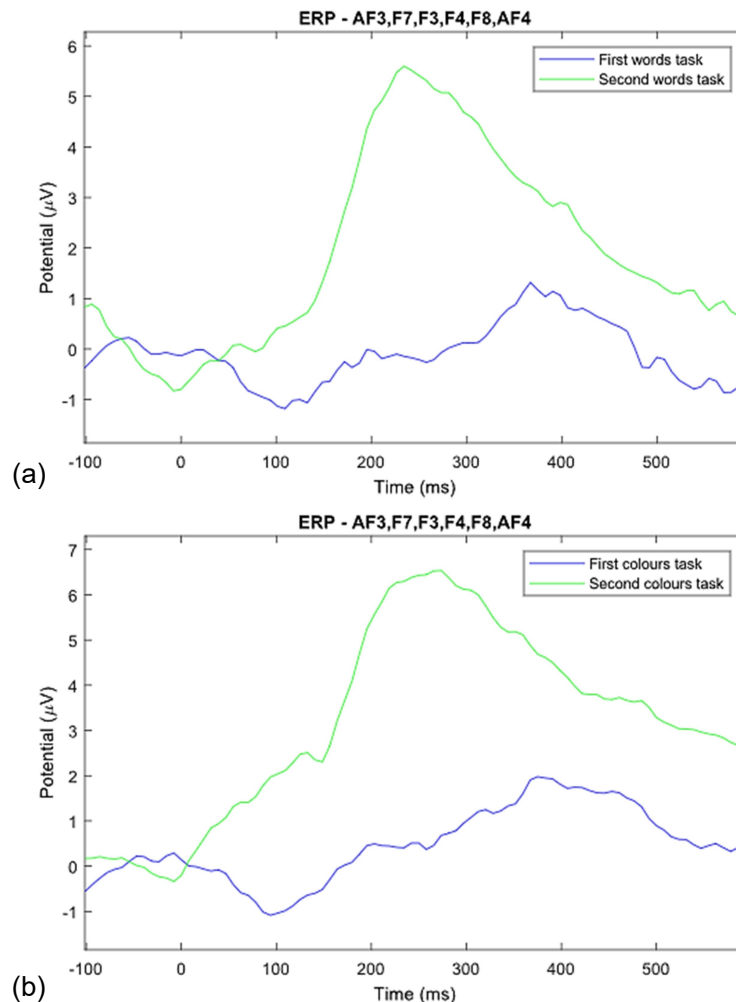
**Figure 23: Stroop words ERPs from the (a) Brain Products (shown for Fz) and (b) Emotiv (shown for the average frontal region) device split into whether the device was the subjects' first or second mode of testing**

Figure 24 show the Stroop colours ERPs for the Brain Products and Emotiv devices, split into the two groups that did the tests with the device first and second (i.e. the subjects' first and second Stroop colours task). These ERPs were also obtained using the original references and sampling rates.



**Figure 24: Stroop colours ERPs from the (a) Brain Products (shown for Fz) and (b) Emotiv (shown for the average frontal region) devices split into whether the device was the subjects' first or second mode of testing**

From Figure 23 and Figure 24, it is observed that ERP peaks for both devices had larger amplitudes if the device was the second mode of testing, and the subjects were thus performing their second Stroop words or colours task. In order to determine whether this can be attributed to practice effects, an overall comparison was done. The data from both devices for the first Stroop words task was compared to the data from both devices for the second Stroop words task. The same was done for the Stroop colours tasks, and the results included in Figure 25.



**Figure 25: ERP comparison between the first and second (a) Stroop words and (b) Stroop colours tasks shown for the averaged frontal region**

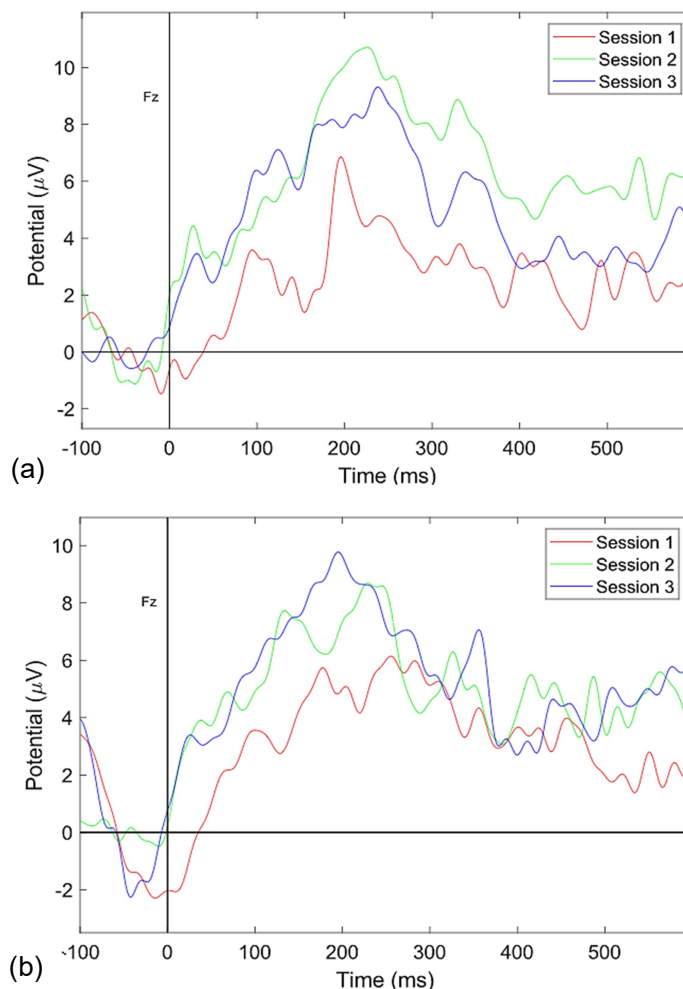
The ERPs in Figure 25 support the idea that the ERP peaks are more distinct, and have larger amplitudes, for the second Stroop task than for the first Stroop task. This may be as a result of practice effects, and should be further investigated with more participants so that higher statistical power can be achieved.

## 5. Concussed participant: Results and preliminary discussion

This chapter includes the results and discussion of the testing of the concussed participant. First, the results of the Stroop words tests are given, followed by the results of the Stroop colours test and the power spectrum analysis of the resting state and tandem data.

### 5.1. ERP analysis of Stroop tasks

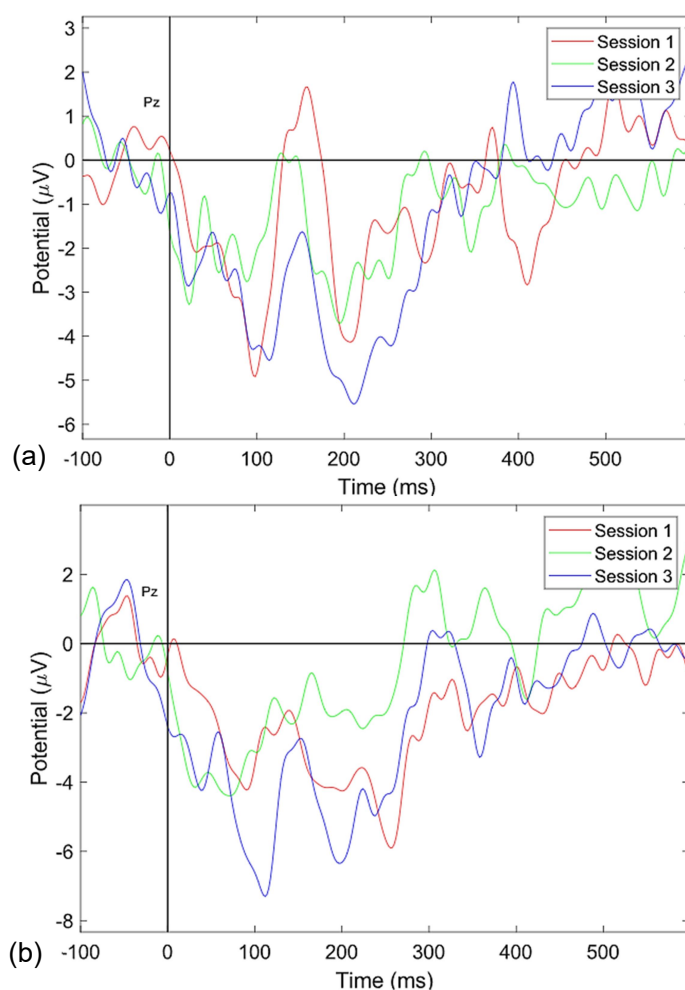
The subject performed one Stroop words and one Stroop colours task at each testing session. As mentioned, Session 1 took place at one day post-concussion, Session 2 took place at 14 days post-concussion and Session 3 took place at 38 days post-concussion. Figure 26 contains comparisons of the ERPs for the Stroop tasks for electrode Fz.



**Figure 26: Stroop (a) words and (b) colours Fz ERPs for the concussed participant**

A study by Broglio et al. (2009) reported post-concussive decrements in ERP amplitudes of the P300 and N200 peaks for previously concussed participants when compared to healthy participants when responding to a novelty oddball task. From Figure 26, we see lower P300 amplitudes in the Session 1 ERPs of both the Stroop colours and Stroop words tasks when compared to Sessions 2 and 3. The concussed participant reported as asymptomatic at two days post-concussion, which accounts for the contradicting Session 2 and Session 3 differences. However, the Stroop colours task has been shown to present greater interference than the Stroop words task (Hotama et al., 2017), thus the P300 peak from Session 2 with a lower amplitude than that from Session 3 may indicate some subtle residual cognitive defects.

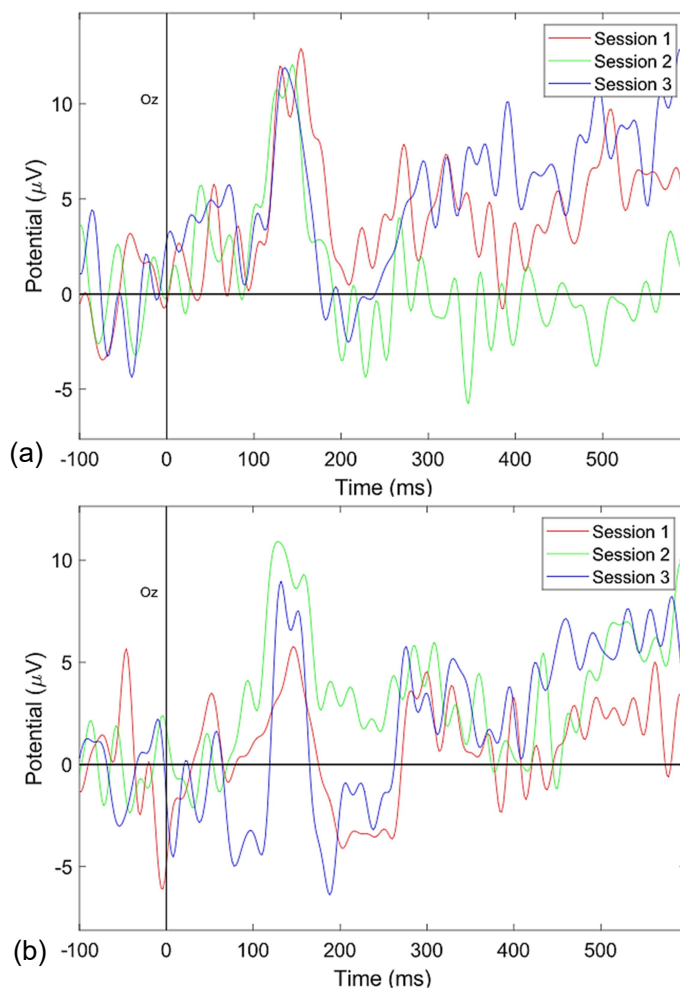
A comparison of the ERPs from the Stroop tasks for electrode Pz is given in Figure 27.



**Figure 27: Stroop (a) words and (b) colours Pz ERPs for the concussed participant**

From Figure 27, we see lower N200 amplitudes for the Session 1 ERPs when compared to the Session 3 ERPs, and the Session 2 N200 peaks have lower amplitudes than both the Session 1 and Session 3 ERPs. This may be attributed to outside factors, such as the high exhaustion level reported by the subject at the time of Session 2. This serves to emphasize the importance of pre-season baseline measures, so that recovery levels can be fully determined and external factors which may influence return-to-play decisions may be minimized. It also emphasizes the importance of retesting concussed players at different points in time to ensure readiness to return to play.

A comparison of the ERPs from the Stroop tasks for electrode Oz is given in Figure 28.



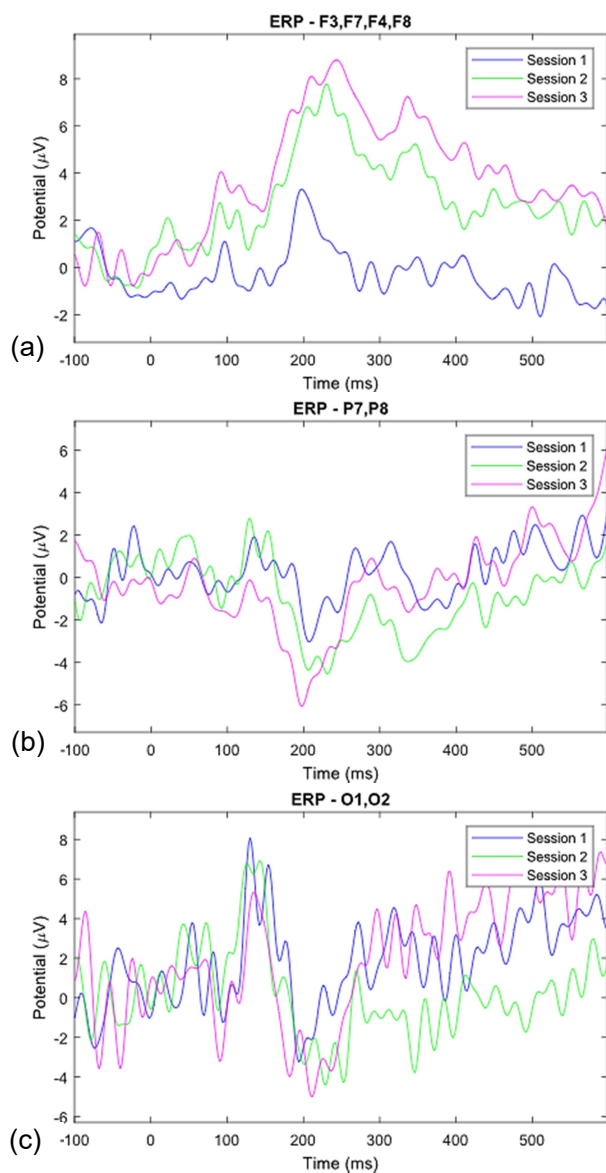
**Figure 28: Stroop (a) words and (b) colours Oz ERPs for the concussed participant**

The results in Figure 28 show similar trends to those in Figure 27, with Session 1 N200 peaks having lower amplitudes than corresponding Session 3 peaks, and Session 2 peaks having lower N200 amplitudes than both Session 1 and Session 3. There also seems to be very distinct positive peak at a latency of 150 ms in the ERPs from all the sessions for both the Stroop words and Stroop colours tasks, which also appears in the Pz ERPs given in Figure 27. These peaks may correspond to the P100 peaks reported by Hudac et al. (2017), which had higher amplitudes in subjects with a history of concussion when compared to a control group. This is also observed as true for the concussed participant in the present study, as the Session 1 P100 peaks have higher amplitudes than the Session 3 peaks for the Pz and Oz electrodes in both the Stroop words and Stroop colours tests.

The data for the concussed participant was re-referenced to an average reference, and the channel ERPs from the Stroop words and Stroop colours tasks were averaged for the frontal region, parietal region and occipital region using the same electrodes as for the healthy participants, with the exception of the frontal region. For the frontal region, the AF3 and AF4 electrodes were not available in the 32-channel montage used, so only the F3, F4, F7 and F8 electrode ERPs were averaged. The results for the Stroop words task are given in Figure 29.

In Figure 29, it is clear that the Session 1 ERPs have the lowest P300 and N200 amplitudes, followed by the Session 2 ERPs and the Session 3 ERPs. The Session 1 ERPs also have the highest P100 amplitudes, followed by the Session 2 and Session 3 ERPs, all of which is consistent with a recovery process supported by previous research. The amplitudes of the ERPs of the sessions are summarised in Table 8.





**Figure 29: Concussed Stroop words average referenced ERPs for (a) frontal, (b) parietal and (c) occipital regions**

**Table 8: Concussed ERP peaks compared to healthy averages for Stroop words [ $\mu\text{V}$ ]**

Peak	Healthy	Session 1	Session 2	Session 3
Frontal P300	6.3	3.3	7.8	8.8
Parietal N200	-4.2	-3.0	-4.5	-6.1
Occipital N200	-4.7	-3.2	-4.4	-5.0

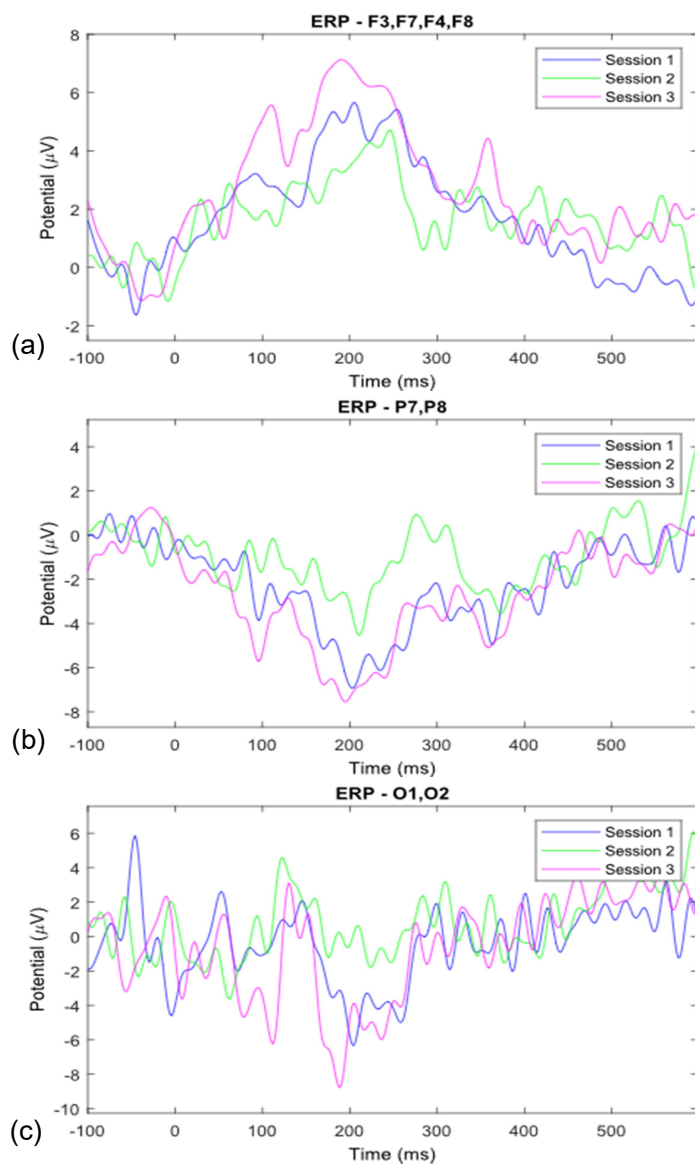
From these peaks it is shown that the concussed participants passed the target obtained from the healthy participants between Sessions 1 and 2 for the frontal and parietal regions and between Sessions 2 and 3 for the occipital region. This reinforces the idea that the proposed protocol is sensitive to showing changes over the recovery period.

The results after re-referencing the data to a common average and averaging the electrode ERPs of the Stroop colours task are given in Figure 30, and the peaks summarised in

Table 9 for comparison to the healthy averages. These results are less conclusive than those of the Stroop words tests, as the ERP peaks for the parietal and occipital regions already have a larger amplitude for Session 1, and the ERPs for the frontal region never reach the target of the healthy average.

Despite the peaks not comparing well to the healthy targets, there are still observable increases in amplitude for all three electrodes from Session 1 to Session 3. The Session 2 peaks have the lowest amplitudes of the three sessions for all three electrodes, which may again be attributed to external factors such as exhaustion, which may have impaired the subject's concentration.

In conclusion for this section, the resulting ERPs have shown shifts in peak amplitude from Session 1 to Session 3 for all graphs plotted which are consistent with differences reported in literature. Session 2 results are inconsistent, possibly due to external factors such as the self-reported exhaustion of the participant. If this Stroop ERP protocol were to be implemented in practice, it should be ensured that readings are taken at different points in time to ensure consistency of resolved symptoms before return-to play decisions are made. The testing sessions should be sufficiently far apart to avoid practice effects skewing the data.



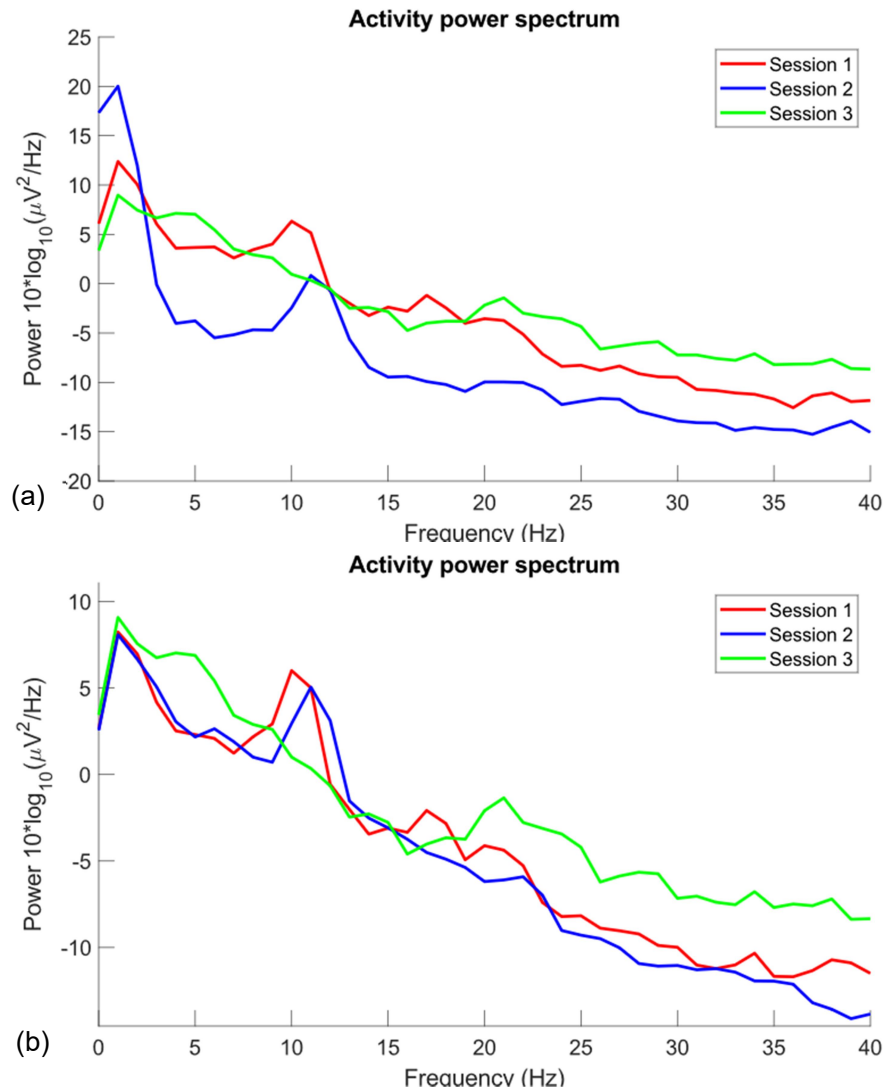
**Figure 30: Concussed Stroop colours average referenced ERPs for (a) frontal, (b) parietal and (c) occipital regions**

**Table 9: Concussed ERP peaks compared to healthy averages for Stroop colours [µV]**

Peak	Healthy	Session 1	Session 2	Session 3
Frontal P300	7.8	5.7	4.7	7.1
Parietal N200	-2.2	-4.5	-6.9	-7.5
Occipital N200	-2.4	-6.3	-1.8	-8.8

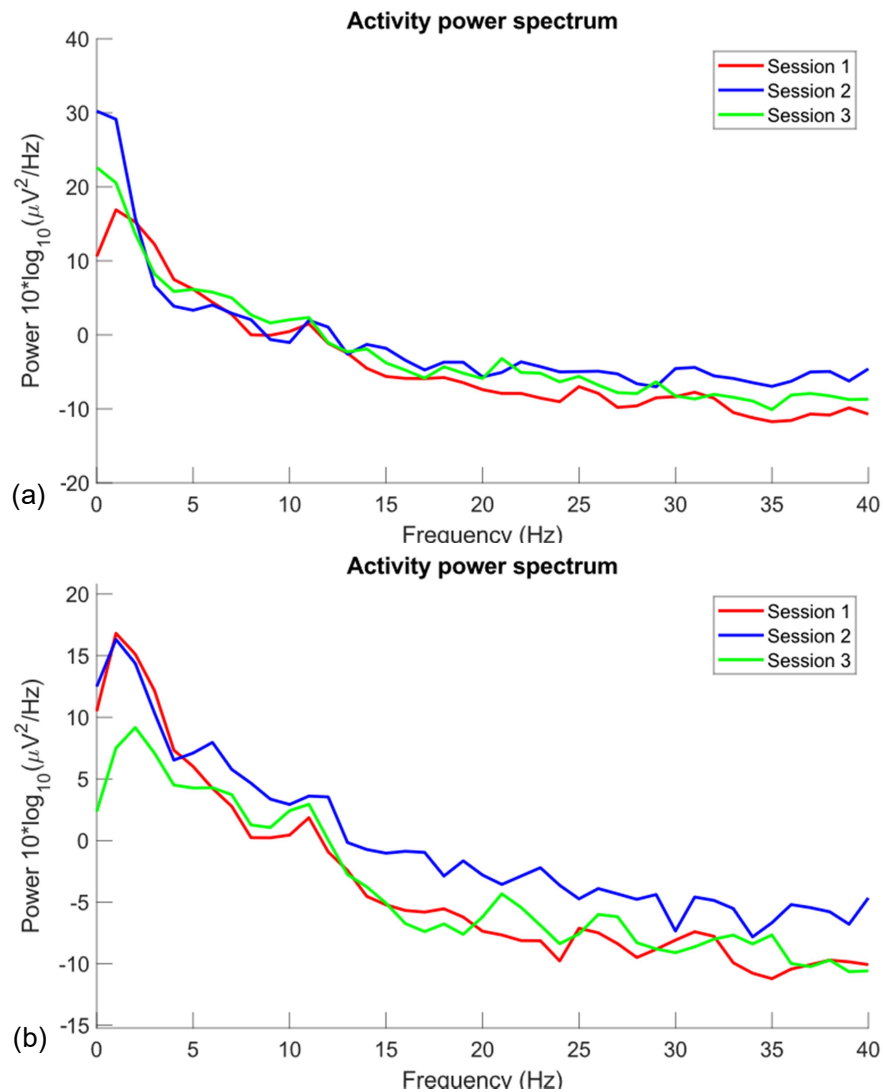
## 5.2. Power spectrum analysis

A power spectrum analysis was conducted using the methods outlined in Chapter 3 on the resting state and tandem stance data obtained from the concussed participant. The results of the power spectrum analysis are represented graphically in Figure 31 for the resting state data and Figure 32 for the tandem stance data. The power spectrums containing the inclusion of all components are given, as well as the spectrums pruned of non-brain components.



**Figure 31: Resting Power Spectrum for (a) original and (b) pruned data**

It is clear from Figure 31 that pruning the components which are not related to brain activity resulted in the power spectrums from Sessions 1 and 2 shifting closer together, while the spectrum obtained from Session 3 remained more or less the same. The power spectrums of Session 1 and 2 follow similar trends across the frequency bands for the pruned spectrums, while the spectrum obtained from Session 3 contains no distinguishable alpha peak. It is possible that this spectrum is still influenced by components related to noise or muscle activity which were overlooked during component rejection or not identified by the ICA.



**Figure 32: Tandem Power Spectrum for (a) original and (b) pruned data**

The pruning of the ICA components obtained for the tandem stance data (Figure 32) resulted in the spectrums shifting closer together, particularly in the delta frequency band. The pruned spectrums for Sessions 1 and 3 seem to follow one another across the frequency range, while the power spectrum for Session 2 follows the same trends at a slightly higher power. Discernable alpha peaks exist for the pruned power spectrums of all three sessions, which indicates that the spectrums are likely comprised of a majority of brain activity components as opposed to components obtained from noise or muscle activity.

A summary of the peak power for each frequency band is included in Table 10 for the resting state data and Table 11 for the tandem stance data. The peaks for both the original and pruned datasets are included.

**Table 10: Concussed resting state power spectrum peaks per frequency band [ $10 \cdot \log_{10}(\mu V^2/Hz)$ ]**

	Delta		Theta		Alpha		Beta	
	Original	Pruned	Original	Pruned	Original	Pruned	Original	Pruned
<b>Session1</b>	12.39	8.24	3.60	2.52	6.33	6.01	-1.18	-2.02
<b>Session2</b>	20.02	8.08	-3.77	3.05	0.83	5.05	-5.63	-1.53
<b>Session3</b>	8.97	8.98	7.13	6.02	2.94	2.16	-1.44	-4.39

**Table 11: Concussed tandem stance power spectrum peaks per frequency band [ $10 \cdot \log_{10}(\mu V^2/Hz)$ ]**

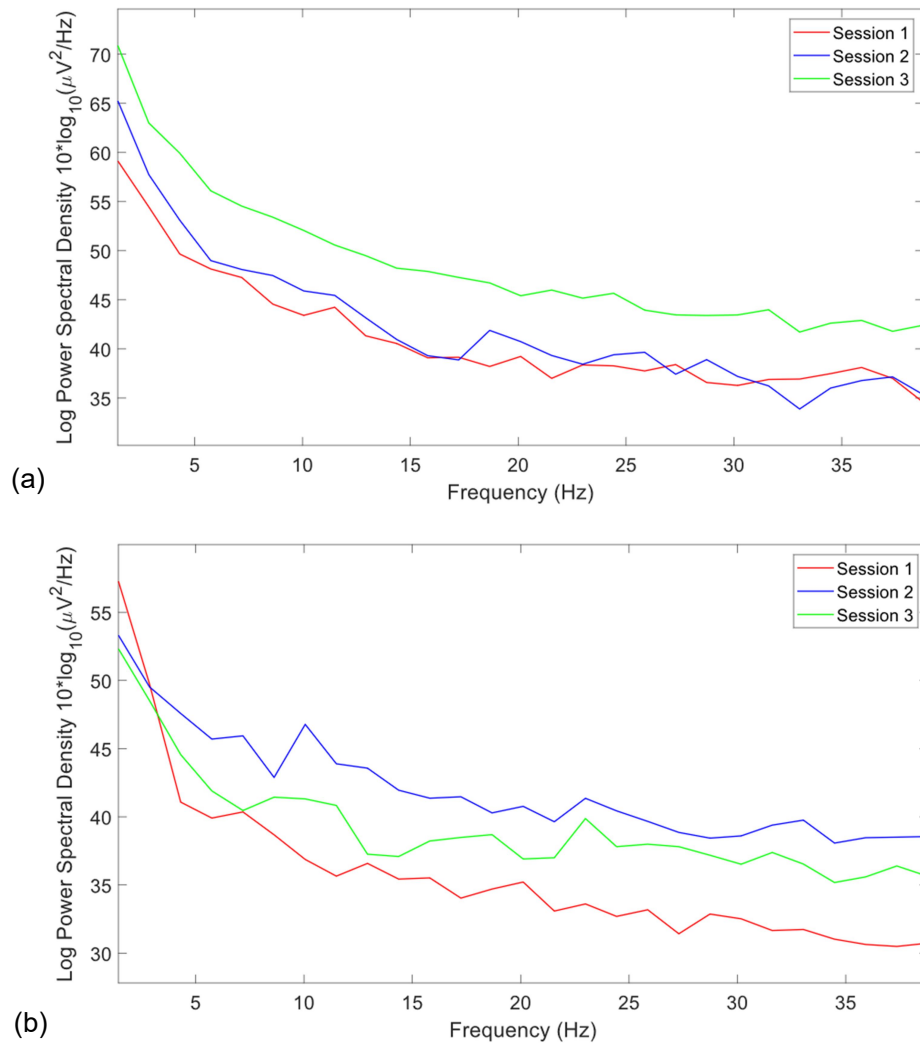
	Delta		Theta		Alpha		Beta	
	Original	Pruned	Original	Pruned	Original	Pruned	Original	Pruned
<b>Session1</b>	16.90	16.81	7.49	7.33	1.49	1.85	-2.47	-2.43
<b>Session2</b>	30.24	16.31	4.03	7.96	2.05	3.61	-1.29	-0.16
<b>Session3</b>	22.63	9.17	6.15	4.50	2.69	2.94	-1.90	-2.76

Previous studies have indicated that concussed groups have lower alpha and beta power when compared to control groups (McCrea et al., 2010). When applying that to this study, there is an expected increase in alpha and beta power moving from Session 1 through Session 2 to Session 3. However, the findings suggest that there is a decrease in alpha power for the pruned resting state data from Session 1 to Session 2, and again from Session 2 to Session 3. The beta power did increase from Session 1 to Session 2, but decreased again from Session 2 to Session 3 to below the Session 1 power. This indicated that the power spectrums cannot be reliably used in this way to determine deficits caused by concussions, but should be further investigated with more concussed subjects to determine the full limitations of the method.

From the power spectrum peaks for the tandem stance data included in Table 11, the expected increase in alpha power is observed from Session 1 to Session 2, followed by a decrease in alpha power from Session 2 to Session 3 to above the Session 1 value. This trend is to some extent consistent with previous literature,

as there is an increase in alpha power from Session 1 to Session 2 and from Session 1 to Session 3. The decrease in alpha power from Session 2 to Session 3 may be attributed to external factors, and should be further investigated with more subjects so that these external factors can be eliminated. There is an increase in beta power for the pruned tandem stance data from Session 1 to Session 2 which is consistent with the trends reported in literature. However, the beta power decreases again from Session 2 to Session 3 to below the beta power of Session 1, which is concerning. This again warrants further investigation with more participants to determine possible reasons for this shift.

The power spectrums for the Stroop colours and words tests are included in Figure 33 (a) and (b) respectively. For the spectra obtained from the different sessions for the Stroop words task, a clear trend can be observed. There is an increase in the overall spectrum from Session 1 to Session 2, and again from Session 2 to Session 3. This is consistent with the notion that concussed participants have decreased power post-concussion, as the power used while performing the Stroop words tasks seems to correlate positively with time since concussion. This is true to some extent for the power spectra of the Stroop colours tests. There is an increase in power used from Session 1 to Session 2 and from Session 1 to Session 3, but there is a decrease in power from Session 2 to Session 3. This may be attributed to residual noise that could not be filtered or removed, or muscle activity which was overlooked in the ICA decomposition or pruning.



**Figure 33: Concussed Stroop (a) words and (b) colours power spectra**

In conclusion for this section, the power spectrum analysis yielded varying results across the different testing modalities. Trends between Session 1 and Session 3 are generally consistent with those in literature, but power spectra from Session 2 seem influenced by external factors and are generally not consistent with literature. The power spectrum analysis for the Stroop words task seems most consistent with findings in literature.



## 6. Discussion

An overall comparison of the Brain Products Brainvision and the Emotiv Epoc showed similar trends in ERP data for the frontal, parietal and occipital regions with decreased amplitudes in both the P300 and N200 peaks for the Emotiv data. There also seemed to be an approximately 200 ms delay in the ERP peaks obtained from the Emotiv data, while the Brain Products peaks seemed to correlate better with the expected peak latencies of 300 ms and 200 ms. This delay in latencies of the Emotiv peaks may be as a result of delays in the sending of markers when recording took place. The markers for the Brain Products device were transferred from a stimulus-producing computer to a recording computer via a trigger cable connected to the Brain Products device itself, while the Emotiv stimuli and recording were done using one device. The sending of markers from the Psychopy stimulus-producing software via a virtual serial port bridge to the Emotiv Xavier Pure recording software may have proved too much for the RAM capacity of the computer, resulting in delays in data transmission. It is suspected that the peaks obtained may actually be the averaged peaks of the following stimuli, corresponding to about a 600 to 700 ms delay (based on the average 800 to 900 ms response time and 200 ms latency delay).

The reduced amplitude of the Emotiv peaks may be attributed to the conductivity of the saline solution when compared to the electrolyte gel used for the Brain Products device. Since the potential differences measured are so small, good conductivity is needed to capture information. A reduced conductivity between the scalp and the electrode would mean an increased resistance for the path travelled by the signal, thereby reducing the sensitivity of the device to detect these potential differences. Another possible reason for this difference in amplitude is the one-size-fits-all nature of the Emotiv device, which could result in incorrect electrode placement when compared to the fitted caps used by the Brain Products device.

The ERP graphs obtained from the from the Emotiv device appeared to contain more noise, while the graphs from the Brain Products device were smoother. This could also be attributed to the conductive properties of the electrolyte gel when compared to the saline solution. The theory that the electrolyte gel is better at conducting the signals is supported by the graphs in Figure 21 and Figure 22, which indicate that the Emotiv data compared better to the Brain Products data if the Emotiv was the subject's second mode of testing.

An interesting finding from Section 4.4 was that P300 peaks seemed to have larger amplitudes for the subjects' second Stroop words/colours tasks than for their first words/colours tasks. This may be attributed to a higher focus of attentional resources during the second Stroop task, as during the first Stroop task the subject may still be getting used to the colour-key associations.

When the P300 capabilities of the Emotiv were investigated previously in comparison to a research grade EEG device, it was found that the Emotiv performed significantly worse (Duvina et al., 2013). This was confirmed by the results of this study, as high data variability resulted in the rejection of the null hypothesis for most conditions. However, since the same general trends were obtained for the averaged ERP comparisons, further research should be

conducted into the sensitivity of the Emotiv device in detecting concussive defects.

Most results of the statistical analysis indicated low statistical power for the study. Dumas-Mallet et al. (2017) indicated that low statistical power is a common occurrence in studies in the biomedical sciences. They reported that approximately 50% of studies have statistical power below 20%, well below the conventional minimum of 80%. It should be noted that in calculating these figures, a level of significance of 5% was used, whereas 10% was used in this study. The level of statistical power is an important consideration in research, therefore further study is recommended with larger sample sizes to confirm the results obtained here.

The results of the concussed participant were mostly consistent with those from previous literature. Lower P300 and N200 peaks were obtained at one day post-concussion than at later stages in the recovery process, which is consistent with the results of Broglio et al. (2009). It is important to note that in the study by Broglio et al., they compared a group with a history of concussions (free from injury at the time) to a control group. Since the trends observed in the data of the concussed participant included in this study move from lower amplitudes to higher amplitudes as time from injury increases, and Broglio et al. found decreased amplitudes in the group with a history of concussion when they reported as free from injury, this supports the notion that concussions may have long-term neurological effects. Broglio went on to explain in another study (Broglio et al., 2011) that these reduced amplitudes may be as a result of reduced attentional resources. Further research should be conducted which include baseline measures and track the participants' recovery process to see when, if ever, full neurological recovery is achieved.

Investigation into the differences between the P300 and N200 peaks between the concussed and healthy participants indicated that the average results of the healthy participants were good indications of a recovery measure for the concussed participant. The concussed participant reported as recovered at 2 days post-injury, which is supported by his ERP peaks passing the amplitude of the peaks of the healthy participants between Session 1 and Session 2. However, changes between Session 2 and Session 3 in several regions of the analysis indicate that the recovery process may still have been on-going.

With only one concussed participant, the conclusions that can be drawn from the data are limited. The results of the participant do seem to indicate that the proposed protocol is sensitive to the detection of post-concussion deficits using the Brain Products device. This should be confirmed with more concussed participants before being tested with a low-cost device such as the Emotiv.

Another possibility for improving the results of the testing on both the healthy and concussed participants would be to increase the number of trials for the Stroop tasks and increase the length of the resting state and tandem stance recordings. This should result in smoother, more reliable ERPs and power spectrums.

In conclusion, the results of the concussed participant preliminarily indicate that the proposed protocol is sensitive to detecting the effects of concussion. In testing the protocol on healthy participants using the Brain Products Brainvision

as a research-grade standard for comparison to the low-cost commercially available Emotiv, it was found that the Emotiv is able to detect the same ERP trends as the Brain Products device. However, the P300 and N200 peaks had reduced amplitudes and an increased latency. The high variability in the data resulted in no statistically significant similarities between the two devices, but statistically significant differences were found in the power spectrums obtained with the two machines.

## 7. Future recommendations

This study investigated the use of the Emotiv Epoc for the use in concussion recovery monitoring using ERPs and power spectrum analyses by comparing it to the Brain Products Brainvision. While similar trends were observed for the two devices, differences in ERP peaks and latencies were observed. A statistical analysis showed low statistical power for many of the observations.

The monitoring of the recovery process of the concussed participant using the outlined methods yielded promising results which warrant further investigation.

Future recommendations for the project are thus:

- Repeat the machine comparison for the promising variables using more participants to ensure a higher statistical power.
- Investigate whether the Emotiv Epoc has sufficient sensitivity to measure changes in ERPs and power spectrums following concussion.
- Investigate normal variability in EEG data for the two systems and whether the systems can be considered reliable in detecting changes in ERPs considering this variability.
- Repeat the testing procedure of the concussed participant on more concussed participants to determine whether the trends observed were unique.
- Include baseline testing on sports players with the potential of obtaining concussions so that if concussions occur, the post-concussion results can be compared to the baseline results to determine when (if ever) the results of the testing return to baseline.
- Determine whether longer resting state and tandem stance recordings will result in more consistent power spectrum results.
- Determine whether including more trials in the Stroop tasks will result in more consistent ERP peak amplitudes.
- Conduct more Stroop tasks to investigate practice effects of the data (including the ERP peaks).
- Investigate whether the use of electrolyte gel with the Emotiv device would increase the quality of recordings.

Much research is still necessary to confirm and build on the findings of this study. This research will aid in building on the body of knowledge in the field of concussion management so that concussions can be better understood and properly managed.

## 8. Conclusion

A study was conducted to investigate a method of monitoring post-concussion changes at the brain functional level to determine when an injured player was ready to return to play. The study included an investigation into whether a low-cost commercially available device could deliver results which were comparable to those of a research-grade device. Also, the recovery of a concussed participant was monitored using a developed protocol, and it was determined that the protocol was sensitive to changes in brain activity post-concussion, which warrants further investigation for confirmation.

The results of the machine comparison indicated that the low-cost Emotiv device was able to detect the same averaged trends as the research-grade Brain Products device, but with lower ERP peaks and a delayed latency. The highly variable nature of the data resulted in no statistically significant similarities being found between the two machines, but statistically significant differences were found in variables of the power spectrum analysis. The results suggest that the Emotiv device is not capable of recording data of the same standard as that of the Brain Products device, but that measuring differences using fewer electrode channels may be a viable option since similar trends are observed across various channels. However, with fewer channels it should be ensured that data quality and consistency is sufficient.

This document introduced the study's background, motivation, objectives and scope, followed by an analysis of the relevant literature. The methodology followed was outlined, including the developed protocol for detecting post-concussion variables in ERP and power spectrum data. The results of the study were included with a preliminary discussion, followed by an overall discussion of the results. Finally, future recommendations were made for other research in the field.

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## Appendix A: Ethical approval letters

The Approval Notice from the Health Research Ethics Committee for this project is included in this Appendix, as well as the Approval with Stipulations for an Amendment submitted.



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Health Research Ethics Committee (HREC)

Approval Notice

New Application

15/02/2018

Project ID :1716

HREC Reference #: S17/10/214

Title: Concussion Assessment Using Electroencephalography (EEG)

Dear Miss Tayla Froneman,

The **Response to Modifications** received on 14/02/2018 14:45 was reviewed by members of the **Health Research Ethics Committee 2 (HREC2)** via **expedited** review procedures on 15/02/2018 and was approved.

Please note the following information about your approved research protocol:

Protocol Approval Period: **This project has approval for 12 months from the date of this letter.**

Please remember to use your **Project ID (1716)** on any documents or correspondence with the HREC concerning your research protocol.

Please note that the HREC has the prerogative and authority to ask further questions, seek additional information, require further modifications, or monitor the conduct of your research and the consent process.

### After Ethical Review

Translation of the informed consent document(s) to the language(s) applicable to your study participants should now be submitted to the HREC.

Please note you can submit your progress report through the online ethics application process, available at: Links Application Form Direct Link and the application should be submitted to the HREC before the year has expired. Please see [Forms and Instructions](#) on our HREC website ([www.sun.ac.za/healthresearchethics](http://www.sun.ac.za/healthresearchethics)) for guidance on how to submit a progress report.

The HREC will then consider the continuation of the project for a further year (if necessary). Annually a number of projects may be selected randomly for an external audit.

### Provincial and City of Cape Town Approval

Please note that for research at a primary or secondary healthcare facility, permission must still be obtained from the relevant authorities (Western Cape Department of Health and/or City Health) to conduct the research as stated in the protocol. Please consult the Western Cape Government website for access to the online Health Research Approval Process, see: <https://www.westerncape.gov.za/general-publication/health-research-approval-process>. Research that will be conducted at any tertiary academic institution requires approval from the relevant hospital manager. Ethics approval is required BEFORE approval can be obtained from these health authorities.

We wish you the best as you conduct your research.

For standard HREC forms and instructions, please visit: [Forms and Instructions](#) on our HREC website <https://applyethics.sun.ac.za/ProjectView/Index/1716>

If you have any questions or need further assistance, please contact the HREC office at 021 938 9677.

Yours sincerely,

Francis Masiye,

HREC Coordinator,

Health Research Ethics Committee 2 (HREC2).

REC-130408-012 (HREC1)-REC-230208-010 (HREC2)

Federal Wide Assurance Number: 00001372  
Office of Human Research Protections (OHRP) Institutional Review Board (IRB) Number:  
IRB0005240 (HREC1)-IRB0005239 (HREC2)

*The Health Research Ethics Committee (HREC) complies with the SA National Health Act No. 61 of 2003 as it pertains to health research. The HREC abides by the ethical norms and principles for research, established by the [World Medical Association \(2013\). Declaration of Helsinki: Ethical Principles for Medical Research Involving Human Subjects](#); the South African Department of Health (2006). [Guidelines for Good Practice in the Conduct of Clinical Trials with Human Participants in South Africa \(2nd edition\)](#); as well as the Department of Health (2015). Ethics in Health Research: Principles, Processes and Structures (2nd edition).*

*The Health Research Ethics Committee reviews research involving human subjects conducted or supported by the Department of Health and Human Services, or other federal departments or agencies that apply the Federal Policy for the Protection of Human Subjects to such research (United States Code of Federal Regulations Title 45 Part 46); and/or clinical investigations regulated by the Food and Drug Administration (FDA) of the Department of Health and Human Services.*



## Approved with Stipulations

### Amendment

20/04/2018

**Project ID:** 1716

**HREC Reference #:** S17/10/214

**Title:** Concussion Assessment Using Electroencephalography (EEG)

Dear Miss Tayla Froneman,

Your amendment request dated 16 April 2018 refers.

The Health Research Ethics Committee (HREC) reviewed and approved the amendments to your study with stipulations.

The following amendments were reviewed and approved with the stipulations below:

1. Revised Protocol Version 1.0 Dated 16 April 2018
2. Revised Informed Consent Form Version 1.0 Dated 16 April 2018
3. Revised Recruitment Flyer Version 1.0 Dated 16 April 2018.

The stipulations of your ethics approval are as follows:

1. A change in the study methodology is perfectly acceptable considering the day-to-day challenges one faces in conducting research. However, the research questions, aims and objectives should be altered to reflect the new questions/aims to be answered. For example, even though the 30 days post-concussion will be considered the 'baseline' – this is not a true baseline but merely an additional time-point and should be reflected as such, especially considering the potential of cumulative concussion.
2. Additionally, the second and third research questions are no longer applicable since there is no baseline to compare these measurements to. Additionally, comparisons between the two different EEG methodologies are not reflected in the research questions/aims/objectives. It is therefore suggested that the study aims/objectives/questions be adapted to answer the new questions that are being posed.
3. Recruitment flyer: Will any individuals with no history of mental illness and no history of concussion be recruited for this study including females and those >23 years of age? If not, please include these requirements in the flyer

Please remember to use your Project ID [1716] on any documents or correspondence with the HREC concerning your research protocol.

Please note that this decision will be ratified at the next HREC full committee meeting. HREC reserves the right to suspend approval and to request changes or clarifications from applicants. The coordinator will notify the applicant (and if applicable, the supervisor) of the changes or suspension within 1 day of receiving the notice of suspension from HREC. HREC has the prerogative and authority to ask further questions, seek additional information, require further modifications, or monitor the conduct of your research and the consent process.

#### After Ethical Review:

Please note you can submit any documentation through the online ethics application process, available at: <https://apply.ethics.sun.ac.za>. Please see [Forms and Instructions](#) on our HREC website for guidance on how to submit any documentation.

#### Provincial and City of Cape Town Approval

Please note that for research at a primary or secondary healthcare facility, permission must still be obtained from the relevant authorities (Western Cape Department of Health and/or City Health) to conduct the research as stated in the protocol. Please consult the Western Cape Government website for access to the online Health Research Approval Process, see: <https://www.westerncape.gov.za/general-publication/health-research-approval-process>. Research that will be conducted at any tertiary academic institution requires approval from the relevant hospital manager. Ethics approval is required BEFORE approval can be obtained from these health authorities.

We wish you the best as you conduct your research.

For standard HREC forms and instructions, please visit: [Forms and Instructions](#) on our HREC website ([www.sun.ac.za/healthresearchethics](http://www.sun.ac.za/healthresearchethics))

If you have any questions or need further assistance, please contact the HREC office at 021 938 9677.

Yours sincerely,

Francis Masiye

HREC Coordinator,

Health Research Ethics Committee 2 (HREC2).

*National Health Research Ethics Council (NHREC) Registration Number:*

*REC-130408-012 (HREC1)-REC-230208-010 (HREC2)*

*Federal Wide Assurance Number: 00001372*

*Office of Human Research Protections (OHRP) Institutional Review Board (IRB) Number:*

*IRB0005240 (HREC1)-IRB0005239 (HREC2)*

*The Health Research Ethics Committee (HREC) complies with the SA National Health Act No. 61 of 2003 as it pertains to health research. The HREC abides by the ethical norms and principles for research, established by the [World Medical Association \(2013\) Declaration of Helsinki: Ethical Principles for Medical Research Involving Human Subjects](#); the South African Department of Health (2006). [Guidelines for Good Practice in the Conduct of Clinical Trials with Human Participants in South Africa \(2nd edition\)](#); as well as the Department of Health (2015). Ethics in Health Research: Principles, Processes and Structures (2nd edition).*

*The Health Research Ethics Committee reviews research involving human subjects conducted or supported by the Department of Health and Human Services, or other federal departments or agencies that apply the Federal Policy for the Protection of Human Subjects to such research (United States Code of Federal Regulations Title 45 Part 46); and/or clinical investigations regulated by the Food and Drug Administration (FDA) of the Department of Health and Human Services.*



## Appendix B: Informed consent form

This Appendix contains a copy of the informed consent form signed by participants.

### PARTICIPANT INFORMATION LEAFLET AND CONSENT FORM

**TITLE OF THE RESEARCH PROJECT:** Concussion Assessment using Electroencephalography (EEG)

**REFERENCE NUMBER:** S17/10/214

**PRINCIPAL INVESTIGATOR:** Tayla Froneman

**ADDRESS:** 8 Honeysuckle, Welgevonden Estate, Stellenbosch

**CONTACT NUMBER:** 0721385091

You are being invited to take part in a research project. Please take some time to read the information presented here, which will explain the details of this project. Please ask the study staff or doctor any questions about any part of this project that you do not fully understand. It is very important that you are fully satisfied that you clearly understand what this research entails and how you could be involved. Also, your participation is entirely voluntary and you are free to decline to participate. If you say no, this will not affect you negatively in any way whatsoever. You are also free to withdraw from the study at any point, even if you do agree to take part.

This study has been approved by the Health Research Ethics Committee at Stellenbosch University and will be conducted according to the ethical guidelines and principles of the international Declaration of Helsinki, South African Guidelines for Good Clinical Practice and the Medical Research Council (MRC) Ethical Guidelines for Research.

#### What is this research study all about?

- *The study will be conducted at the Neromechanics Lab in Stellenbosch. It will involve 30 healthy participants to help with machine comparison, and between ten and twenty concussed participants to help with the recovery monitoring testing.*
- *The project aims to investigate whether the brain heals more slowly after a concussion than your symptoms would have you believe. It is believed that your brain can still show some abnormalities after all the visible symptoms of the concussion have gone away. By determining when your brain has really recovered from an injury, the study aims to avoid repeated concussions while the brain is still injured, as this can lead to very serious problems.*
- *The lightweight EEG measuring device will be placed on your head and a gel will be applied to the electrodes using a blunt needle. You will be asked to first sit still in a chair for 60 seconds with your eyes closed while we take readings in this "resting state". After this, we will ask you to stand in a "tandem stance" for 28 seconds while we take more readings. This stance involves standing up straight with one foot aligned directly behind the other. The toe of the back foot must touch the heel of the front foot. You can then sit back down and will perform a*

*stroop test while EEG measurements are taken. The stroop test involves reading 36 words printed in various colours out loud, and also naming the colours that the words are printed in. You should correct any mistakes made as you go. The device will then be removed from your head and the testing will be complete. If you are one of the participants helping with machine comparison (i.e. you do not have a concussion), then a second device (which does not need to be gelled) will be placed on your head. You will have to repeat the procedure just explained.*

- *One testing session will take at most 45 minutes for the concussed participants and one hour for the machine comparison participants. Participating in the study will involve only one session if you are a healthy participant and four sessions (at one, seven, fourteen and thirty days post-concussion) if you are a concussed participant.*

#### **Why have you been invited to participate?**

- *You have been invited to participate in this study as you fall in one of two categories:*
  - *You are healthy and have no history of mental illness and are thus considered a suitable candidate for machine comparison testing.*
  - *You have obtained a concussion in the last 24 hours and can be scheduled for a first round of testing. New measurements will then be taken at 7, 14 and 30 days after your concussion to determine the rate at which your brain heals. You should have no open wounds on your head or lower body injuries which may affect your balance.*

#### **What will your responsibilities be?**

- *You have to perform the procedures outlined above for a once-off measure (if applicable), or at one, seven, fourteen and thirty days post-concussion (should you have a concussion).*

#### **Will you benefit from taking part in this research?**

- *By participating in this research you can help us determine when players are fully healed from concussions. This may influence future return-to-play decisions. If a player is not fully recovered when he/she returns to play, they stand a greater chance of obtaining another concussion. The effects of this may include serious long-term effects or even death. It is thus very important to know when players have recovered from concussions.*

#### **Are there any risks involved in your taking part in this research?**

- *There are very few risks involved in this research. A small amount of activity is involved in the balance test and may involve some risk of tripping, but the project can be considered low-risk overall.*

**If you do not agree to take part, what alternatives do you have?**

- *If you do not take part in the study, you are free to pursue the normal course of treatment and follow-ups with physicians at Campus Health Services (or another physician of your choice) in the event of a concussion. It is important to note that the normal course of treatment will continue irrespective of whether you participate in the study or not.*

**Who will have access to your medical records?**

- *All data acquired will be stored and processed at a later stage. Access to the data will be limited to the primary investigators, and during reporting your identity will be kept confidential. Data will be stored on a hard drive, and backed up in a secure online folder. The hard drive will be locked in an office to which only the primary investigators have access, and the online folder will be password protected. The password will only be known by the primary investigators.*
- *The data that is stored will be split into a personal profile (name, age, gender, number of previous diagnosed concussions) which will be assigned a subject number in the study, and EEG related data that is collected during testing. These will be stored separately in the manner described in the point above to help protect your anonymity.*
- *The results of the study will be published in a thesis, but all participants' names will be kept confidential.*

**What will happen in the unlikely event of some form injury occurring as a direct result of your taking part in this research study?**

- *The doctors at Campus Health Services will treat you in the unlikely event of an injury.*

**Will you be paid to take part in this study and are there any costs involved?**

No, you will not be paid to take part in the study, but your transport costs will be covered for each study visit. There will be no costs involved for you, if you do take part. Only transport costs on campus will be covered (e.g. from your residence/ last class) at R1.50 per kilometre up to a maximum of R7.25 for the return trip (campus extends at most 2km from Coetzenberg).

**Is there anything else that you should know or do?**

- You can contact Dr Vivier at tel 021 808 3496 if you have any further queries or encounter any problems.
- You can contact the Health Research Ethics Committee at 021-938 9207 if you have any concerns or complaints that have not been adequately addressed by your study doctor.
- You will receive a copy of this information and consent form for your own records.



### Declaration by participant

By signing below, I ..... **agree to take part in a**  
research study entitled *Concussion Assessment using Electroencephalography (EEG)*

I declare that:

- I have read or had read to me this information and consent form and it is written in a language with which I am fluent and comfortable.
- I have had a chance to ask questions and all my questions have been adequately answered.
- I understand that taking part in this study is voluntary and I have not been pressurised to take part.
- I may choose to leave the study at any time and will not be penalised or prejudiced in any way.
- I may be asked to leave the study before it has finished, if the study doctor or researcher feels it is in my best interests, or if I do not follow the study plan, as agreed to.

Signed at (place) ..... **on (date)** .....

.....  
Signature of participant

.....  
Signature of witness

### Declaration by investigator

I (name) ..... **declare** that:

- ~~I explained the information in this document to~~ .....
- I encouraged him/her to ask questions and took adequate time to answer them.
- I am satisfied that he/she adequately understands all aspects of the research, as discussed above
- I did/did not use an interpreter. *(If an interpreter is used then the interpreter must sign the declaration below.*

Signed at (place) ..... **on (date)** .....

.....  
Signature of investigator

.....  
Signature of witness

### Declaration by interpreter

I (name) ..... declare that

- I assisted the investigator (name) ..... to explain the information in this document to (name of participant) ..... using the language medium of Afrikaans/Xhosa.
- We encouraged him/her to ask questions and took adequate time to answer them.
- I conveyed a factually correct version of what was related to me.
- I am satisfied that the participant fully understands the content of this informed consent document and has had all his/her question satisfactorily answered.

Signed at (place) ..... on (date) .....

.....  
Signature of interpreter

.....  
Signature of witness

## Appendix C: Recruitment flyer

This Appendix includes an example of a flyer used to recruit concussed participants.



### Help needed with concussion research



The Biomedical Engineering Department, Stellenbosch University, is looking for people to participate in a research study which investigates the recovery time for concussions using an EEG machine. Your data will be evaluated by a neurologist to determine the seriousness of your injury and to ensure that other intervention is not necessary.

#### To take part in this study you should meet the following criteria:

- Be a male rugby player between the ages of 18 and 23 who has obtained a concussion within the last 48 hours
- Be currently playing for a rugby team in Stellenbosch and registered with Maties Rugby Club

#### What will be done?

- You will report to the Neuromechanics Lab at Coetzenburg as soon as possible for a full study explanation before being required to sign a form stating that you consent to participate.
- You will be asked to perform tasks which include sitting, balancing and naming words/colours. This testing will not take more than 30 minutes.
- You will be asked to return for testing at 7, 14 and 30 days post-concussion.
- Lifts can be arranged to the lab for all sessions.

This study has been approved by the Human Research Ethics Committee (S17/10/214) and Maties Sport. It is done with the support of Campus Health Services and the Institute of Sport and Exercise Medicine (ISEM). For more information, please contact Tayla Froneman at 0721385091 or [17710383@sun.ac.za](mailto:17710383@sun.ac.za).



keeping US healthy



## Appendix D: Event Related Potential peaks

The ERP peaks obtained from the individual graphs of the healthy participants are given in Table 1.

**Table 12: Individual ERP peaks [ $\mu$ V]**

Subject	Brain Products						Emotiv					
	Words			Colours			Words			Colours		
	F	P	O	F	P	O	F	P	O	F	P	O
1	-3.3	0.4	2.6	2.3	-4.6	-1.4	1.8	-2.5	-4.5	-1.2	-1.2	4.2
2	3.4	-2.4	-1.6	2.5	0.0	-0.7	2.4	-4.5	-4.5	-0.9	-1.8	-1.5
3	1.0	1.7	0.7	49.9	35.9	34.6	-2.1	0.8	1.8	-1.7	1.4	0.1
4	4.7	0.4	-2.2	5.2	-0.9	-3.1	1.0	-1.8	-1.3	-0.4	-3.1	1.4
5	4.1	-2.9	-4.0	3.0	-1.4	-2.1	2.2	-2.2	-4.4	2.9	-1.7	-5.8
6	4.1	-2.0	-3.7	5.6	-3.4	-6.2	4.5	-2.4	-6.2	4.3	-2.1	-6.8
7	7.7	-1.8	-3.7	5.9	-4.9	-0.5	1.9	-2.0	-3.6	0.1	-1.1	-1.5
8	2.7	-2.1	-1.1	6.9	-3.8	-2.6	14.4	-10.4	-17.6	3.1	-4.4	-4.8
9	7.5	-4.7	-3.6	7.2	-6.5	-5.5	0.6	0.6	-1.9	-0.7	-1.0	1.7
10	3.1	-5.6	-5.5	1.5	-5.4	-2.7	2.7	-5.0	-5.2	0.1	-1.2	-1.5
11	3.7	-2.1	-5.1	1.6	-2.5	-3.6	-3.9	5.7	6.2	-1.7	1.8	4.9
12	19.6	-14.7	-14.5	10.9	-10.5	-10.3	-5.9	3.0	1.0	1.0	-2.2	-1.4
13	5.1	-2.5	-4.8	2.7	-3.1	-2.7	1.3	-2.3	-3.4	-0.1	-8.9	-3.0
14	70.1	-45.9	-47.1	58.1	-37.7	-41.9	25.2	-24.6	-28.8	13.8	-15.0	-14.9
15	2.2	-2.3	-4.9	5.4	-3.5	-5.5	0.1	-1.0	-0.4	2.2	-4.7	-4.2
16	6.4	-6.9	-7.2	5.6	-6.3	-6.6	2.0	-0.8	-2.3	4.6	-4.7	-6.0
17	3.8	-3.8	-6.3	1.3	-2.7	-3.0	2.1	-4.2	-3.4	1.5	-1.9	-2.6
18	-	-	-	6.9	-6.8	-7.3	-	-	-	3.0	-0.9	-7.3
19	0.3	-1.0	-1.7	4.8	-4.4	-2.9	1.6	-1.9	-0.4	0.7	-2.6	-1.6
20	11.7	-6.8	-7.7	10.0	-2.8	-1.5	2.8	-2.0	-3.5	3.3	-1.1	-3.1
21	0.1	-3.9	-4.3	5.3	-3.3	-3.6	2.3	-1.5	-2.6	4.8	-6.7	-7.6
22	3.9	-0.4	-1.2	4.2	-1.9	-2.4	-1.9	0.9	1.1	4.3	-5.3	-6.5
Mean	7.7	-5.2	-6.0	9.4	-3.7	-3.7	2.6	-2.8	-4.0	2.0	-3.1	-3.1

Key:

F = Frontal region; P = Parietal region; O = Occipital region

## Appendix E: Power spectrum peaks

The peaks obtained from the power spectrums for the different frequency bands for the resting state and tandem stance data of the healthy participants are given in Table 13 and Table 14 respectively.

**Table 13: Resting state power spectrum frequency band peaks**  
[ $10 \cdot \log_{10}(\mu V^2/Hz)$ ]

Subject Number	Brain Products				Emotiv			
	Delta (1-4 Hz)	Theta (4-8 Hz)	Alpha (8-13 Hz)	Beta (13-30 Hz)	Delta (1-4 Hz)	Theta (4-8 Hz)	Alpha (8-13 Hz)	Beta (13-30 Hz)
1	11.55	-5.85	-6.01	-10.21	24.32	7.20	5.24	-3.45
2	8.63	-4.15	-1.87	-5.06	23.97	21.04	15.88	13.65
3	17.96	6.57	6.14	-3.75	13.71	7.51	7.51	-4.60
4	10.96	11.88	14.83	1.65	26.19	6.54	4.46	-2.66
5	1.78	-2.07	6.01	-3.98	18.94	3.31	6.98	-3.03
6	14.34	2.55	5.56	-4.49	18.42	10.34	3.90	-0.45
7	1.23	-1.91	7.13	-6.25	26.45	21.80	16.84	19.92
8	2.75	-4.53	9.96	-0.54	15.87	7.56	7.97	-0.00
9	9.05	-1.24	-2.26	-8.51	18.95	10.94	2.55	-6.08
10	4.81	-1.54	-1.70	-5.95	32.66	29.54	22.17	18.68
11	2.34	1.76	2.17	-1.68	12.47	3.30	2.03	-3.09
12	3.93	2.29	12.78	-5.23	22.59	6.50	0.35	-8.00
13	5.59	0.31	8.33	-3.16	28.10	9.01	4.26	-4.53
14	5.67	4.84	11.61	-3.06	6.02	-0.29	8.11	-2.97
15	12.40	4.32	11.36	-1.94	5.13	-1.17	8.24	-0.83
16	4.43	-2.48	2.82	-6.74	18.73	2.79	-1.32	-6.77
17	1.90	2.55	6.65	-3.84	35.60	25.60	19.39	14.71
18	10.14	-1.16	-2.36	-6.51	10.36	1.56	-0.48	-2.89
19	9.99	2.52	9.81	-4.65	22.30	9.11	7.02	-4.58
20	10.45	-0.97	-0.15	-3.71	26.23	14.25	9.80	3.76
21	8.85	8.37	12.54	-4.08	8.21	5.52	11.04	-4.62
22	6.43	0.46	8.23	-1.33	9.16	1.25	11.04	0.24



**Table 14: Tandem stance power spectrum frequency band peaks  
[ $10 \cdot \log_{10}(\mu V^2/Hz)$ ]**

Subject Number	BrainProducts				Emotiv			
	Delta (1-4 Hz)	Theta (4-8 Hz)	Alpha (8-13 Hz)	Beta (13-30 Hz)	Delta (1-4 Hz)	Theta (4-8 Hz)	Alpha (8-13 Hz)	Beta (13-30 Hz)
1	19.71	-4.49	-5.64	-8.44	14.87	3.58	6.62	-1.40
2	2.43	-2.82	-1.77	-3.78	18.10	5.25	-0.51	-6.68
3	46.72	25.66	15.51	11.15	17.64	3.61	1.40	-5.93
4	7.28	9.33	15.70	1.33	26.60	7.53	1.76	-7.55
5	5.57	-1.18	3.30	-2.49	11.24	2.26	1.11	-2.75
6	22.35	10.71	1.70	-4.45	23.75	4.54	2.91	-0.80
7	3.89	-0.47	-0.14	-5.55	23.99	4.54	-1.74	-4.34
8	13.09	0.64	7.79	-2.41	16.91	3.94	-0.05	-4.99
9	14.34	6.85	-2.66	-6.33	20.21	19.29	15.26	9.14
10	10.96	0.46	-4.71	-4.64	28.64	6.57	-1.64	-8.73
11	23.85	10.01	-0.31	-4.80	27.14	9.29	0.34	-2.12
12	5.92	-0.54	6.93	-7.24	17.27	3.07	4.05	-5.32
13	5.40	-2.32	7.36	-2.31	27.22	12.89	5.36	3.09
14	8.52	2.74	9.30	-4.52	34.20	14.84	2.48	-4.85
15	7.97	0.99	10.67	-1.10	31.83	22.73	14.53	4.00
16	10.62	-3.96	2.87	-7.88	18.27	0.73	-1.97	-7.40
17	2.60	-1.15	2.83	-3.07	8.60	-1.31	3.77	-1.81
18	12.42	0.40	-4.81	-7.85	25.30	9.86	2.64	-2.48
19	33.36	-0.77	1.60	-6.60	27.29	4.73	4.38	-1.42
20	19.52	2.97	-0.08	-3.89	30.24	5.57	-1.46	-3.78
21	8.38	5.11	13.87	-4.11	8.78	3.45	11.05	-5.16
22	1.15	-2.31	7.62	-0.16	3.17	-1.66	10.64	0.78

## Appendix F: Response times and number correct

The number of correct responses per participant per Stroop task are given in Table 15, while the average response time per subject per task is given in Table 16.

**Table 15: Numbers of correct responses (out of 36)**

Subject Number	Machine based breakdown				Task based breakdown			
	Brain Products		Emotiv		Stroop words		Colours	
	Words	Colours	Words	Colours	First task	Second task	First task	Second task
1	33	33	33	35	33	33	33	35
2	36	34	36	35	36	36	35	34
3	36	36	35	34	35	36	34	36
4	36	35	35	36	36	35	35	36
5	35	35	36	33	35	36	35	33
6	36	35	35	35	36	35	35	35
7	36	36	36	35	36	36	35	36
8	36	36	35	36	36	35	36	36
9	36	36	35	36	35	36	36	36
10	36	35	34	36	36	34	35	36
11	36	36	35	36	35	36	36	36
12	35	36	35	36	35	35	36	36
13	36	36	36	35	36	36	36	35
14	36	36	36	34	36	36	34	36
15	36	36	36	36	36	36	36	36
16	36	35	36	36	36	36	36	35
17	34	36	36	36	34	36	36	36
18	36	36	36	36	36	36	36	36
19	35	36	35	35	35	35	36	35
20	35	36	34	36	34	35	36	36
21	36	36	36	36	36	36	36	36
22	36	35	35	35	35	36	35	35
Total	783	781	776	778	778	781	778	781
Average	35.59	35.5	35.27	35.36	35.36	35.5	35.36	35.5

**Table 16: Average response time per subject per task [s]**

Subject	Stroop words		Stroop colours	
	First task	Second task	First task	Second task
1	0.671	0.643	0.584	0.688
2	0.715	0.756	0.963	0.759
3	0.658	0.797	0.739	0.745
4	0.794	0.869	1.069	0.919
5	0.743	0.719	0.815	0.816
6	0.817	0.784	0.986	0.912
7	0.903	1.207	0.959	1.184
8	1.091	0.734	0.979	0.892
9	0.580	0.513	0.492	0.448
10	0.905	0.849	1.078	0.949
11	0.764	0.878	0.607	0.837
12	0.706	0.714	0.957	0.787
13	0.597	0.687	1.002	0.747
14	1.331	1.207	1.222	0.984
15	1.606	0.987	1.329	1.103
16	0.758	0.765	0.831	0.926
17	1.066	1.113	1.601	1.152
18	0.892	0.967	1.071	0.813
19	0.720	0.755	0.649	0.866
20	0.840	0.938	1.709	1.350
21	0.713	0.618	0.796	0.926
22	1.175	0.863	0.816	1.326
<b>Average</b>	0.866	0.835	0.980	0.915